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## Section of the History of Medicine

President—Sir WALTER LANGDON-BROWN, M.D.

[October 4, 1939]

### W. H. Gaskell and the Cambridge Medical School

#### PRESIDENT'S ADDRESS

By Sir WALTER LANGDON-BROWN, M.D.

THE textbooks of my schooldays seemed designed to give the impression that history ended in 1815 with the battle of Waterloo; after that it was only politics. I hope your views on the history of medicine are not thus limited, for while appreciating deeply the honour you have done me in electing me your President in succession to so many distinguished scholars, I feel an amateur in the presence of experts on the medical history of the distant past. For that among other reasons I have chosen to speak on the recent past. Another reason that impels me is that there cannot be many practising now who actually worked under Gaskell while he was still engaged in his great researches. Those of us who remain faithful to his memory must feel it laid upon them almost as a duty to recall to men's minds the enormous influence of those researches on the subsequent progress of medicine—an influence so widely spread that passing into the general stream of medical thought, its origin is too often forgotten. I hope to be able to show not only the beautifully logical sequence of those researches, but also how they have influenced our conceptions of cardiology, endocrinology, and diseases of the autonomic nervous system, although they were carried out by one whose interests were remote from clinical medicine.

The conjunction in my title may puzzle some of my hearers, who may ask, did Gaskell play an important part in the rise of the Cambridge Medical School? The great bulk of the medical students never encountered him, for he only lectured and demonstrated to advanced classes, which were never large. Any value which this address may have is because I have first-hand knowledge of exactly what part he *did* play and of its real importance. But firstly let us consider what went before.

Dr. Arnold Chaplin has enriched our *Proceedings* with a valuable account of Medical education at Oxford and Cambridge between 1500 and 1850. The record is not very creditable to those ancient seats of learning. Despite the establishment of lectureships by Linacre and then of Regius Professorships by Henry VIII, despite the introduction of anatomical demonstrations by Caius at Cambridge, medical education did not flourish there. Glisson, one of the best known of the Regius Professors, did undoubtedly give an impetus to the study of medicine and anatomy though he was an absentee repeatedly during his forty-one years' tenure of the

Chair. A Chair in Chemistry was established in 1703 and Francis Vigani was appointed to it. His drug chest is still in existence and its contents have been admirably described by Alderman Saville Peck, recently Mayor of Cambridge. In 1705 Richard Bentley, the turbulent Master of Trinity, had a laboratory fitted up for Vigani in the College, largely I fear to thwart his Fellows who were opposed to scientific studies. Here it was that Stephen Hales, after a sound training in Newtonian physics, went on to experiments in physiology which later culminated in his classical observations on blood-pressure. Until 1707 Anatomy was taught by the Regius Professor, but in that year George Rolfe was appointed Professor of Anatomy. It is sad to relate that he was deprived of his office in 1728 for repeated absence. No wonder the school did not flourish! In my student days there was a circular anatomical theatre which so closely resembled that in which Harvey attended demonstrations in Padua that I imagined it was modelled on it for the same purpose. I was therefore surprised to find that it was originally designed for the use of the University Press, a purpose for which it was found quite unsuitable, and was therefore handed over in 1716 to the Professors of Anatomy and Chemistry. In 1833 it was removed to the New Museums in Downing Street where it remained till 1903, to be succeeded by the worst-designed theatre I have ever seen, although its architect was the then Professor of Fine Art. This came to an inglorious and much to be desired end in 1933 during my tenure of the Regius Chair.

It was not until the Prince Consort became Chancellor of the University that the Natural Sciences Tripos was established against considerable opposition, and the first examination for this was held in 1851, the year after Dr. Arnold Chaplin's history ends. Four candidates were placed in the First Class, two in the Second. It is interesting to note that this short list was headed by G. D. Liveing, afterwards Professor of Chemistry for forty-seven years. After his retirement in 1908 he lived on to the age of 97 when his still active life was brought to an end by a Newnham student on a "push bike" who ran over him and fractured his femur. Yet I believe he had favoured the admission of women to the University!

The great triumvirate that created the modern medical school at Cambridge consisted of George Paget, George Humphry and Michael Foster, but it was an earlier predecessor of mine, *John Havilland*, who by his energy and sagacity laid the foundations in 1829, and entirely remodelled the curriculum. When George Paget became Regius in 1872 he vigorously put Havilland's plans into execution. The general stagnation still existing, despite Havilland's efforts, is shown by the fact that in 1870 only 2 M.D. and 7 M.B. degrees were conferred; whereas in my last year of office there were 604 medical students in residence and nearly as many at the hospitals.

I just remember seeing *Sir George Paget* in his old age, a frail but distinguished figure. The Cambridge School owes much to his prophetic vision and untiring effort, just as my other school, St. Bartholomew's, is greatly indebted to Sir James Paget. The effect of these two brothers on medical education has been profound and far-reaching. They have been compared with another great pair of brothers, also surgeon and physician respectively, John and William Hunter. It was on the advice of the Paget brothers that *George Murray Humphry* was brought to Cambridge. A Suffolk man, he was apprenticed at the early age of 16 to the well-known surgeon John Green Crosse at Norwich, and subsequently entered St. Bartholomew's Hospital, where he came under the influence of the famous Peter Mere Latham, whose emphatic style of teaching he adopted. On October 31, 1842, when only 22 years of age, he was elected surgeon to Addenbrooke's, a post he held for fifty-two years. In less than two months he had obtained permission to deliver clinical lectures there. From 1848 he was responsible for the teaching of anatomy, becoming Professor in 1866. His energy was terrific. In addition to teaching surgery and anatomy, he found time from the demands of a busy practice to pour out a constant

stream of papers on anatomical, pathological and surgical subjects, which secured his election as an F.R.S. in 1859. His ambition was to make Cambridge a complete school of medicine, and Addenbrooke's Hospital was largely rebuilt from plans he prepared. He established a hostel for medical students to enable those of small means to come to the University for the whole of their training. But his very success in drawing students made such a scheme impracticable. There was not, and cannot be, clinical material available for a large number of students, although



*W. H. Gaskell*

FIG. 1.—(From "The Cambridge Medical School", by Sir Humphry Rolleston, Bart., G.C.V.O.) (Block kindly lent by the Cambridge University Press.)

Addenbrooke's is far better equipped for the task now than then. In 1883 he resigned the Professorship of Anatomy and offered to become Professor of Surgery without stipend, a post he held until his death in 1896 at the age of 76. One can see him now, his glittering hawk-like eye, ready to pounce on ignorance and sloth, but equally ready to help. A born teacher, he has been described as "magnetic and Socratic"; innumerable anecdotes cluster round his name. One must suffice. "On one occasion a student not remarkable for ability somewhat unexpectedly answered a question correctly, and followed it up by the unwise comment, 'You seem surprised, Professor', only to be discomfited by the prompt retort, 'So was Balaam when his ass spoke'."

Humphry keenly advocated the establishment of a Chair of Physiology, but there

were delays. Here again, as in 1705, Trinity College came to the rescue largely owing to the efforts of Coutts-Trotter, and on Huxley's recommendation brought *Michael Foster* from University College, London, as Praelector and Fellow in 1870. No more fortunate choice could have been made. Although at first his only laboratory was the corner of the Philosophical Library, he rapidly gathered a brilliant band of students round him. *Walter Holbrook Gaskell*, who had graduated as a Wrangler, seeing a notice of a lecture to be given by Foster, strolled in to hear it. That determined his future career with enormous consequences to English physiology and medicine. *J. N. Langley* entered St. John's College the year after Foster's arrival, and after gaining a first class in the Natural Sciences Tripos subsequently became his demonstrator. In 1903, he succeeded Sir Michael Foster in the Professorship which had been established in 1883. Gaskell said of Foster "He was a discoverer of men rather than of facts, and he worked for rather than at physiology". That he had enormous influence in determining the careers of his followers, is manifest; he was a supreme judge of men. This triumvirate of Paget, Humphry and Foster was the controlling factor in ensuring success, but we must not forget the part also played by *Alexander Macalister*. A great anatomist, he took all learning for his province.

And now, the failure at the time of the Renaissance to establish a living school of medicine in Cambridge was at length to be gloriously redeemed. I was not there to see the springtide which followed the long winter, for when I entered in 1889, it was already early summer, but from my teachers I heard much of that swift flowering.

In my time the three University Lecturers under Foster were Gaskell, Langley, and Sheridan Lea. The last named was a good chemical physiologist, but his health broke down and he retired fairly early in life. The Demonstrators were Dr. Lewis Shore who until quite recently played an active rôle in the department as Lecturer, and W. B. Hardy, afterwards Sir William, a brilliant, vital creature who made a great success in three different capacities. It is told of him that the biologists and the physicists simultaneously recommended him for a Royal Medal at the Royal Society for his researches in their particular subjects. His work as Head of the Low Temperature Station was of great value to Empire transport trade. He was also one of the most skilled and intrepid of amateur yachtsmen. A friend said to me that no one really knew Hardy thoroughly who had not seen him handling a small yacht in a stiff gale. I never enjoyed that doubtful pleasure, but I was fascinated by his versatility and vigorous personality. To this staff were added shortly afterwards—H. K. Anderson, afterwards Sir Hugh, who became such an influential University administrator as Master of Caius College, and W. H. R. Rivers whose work in building up a biological approach to a rational psychotherapy still has a great effect, seventeen years after his death. Surely it has rarely happened that any scientific department has had so many brilliant men on its staff at the same time. Yes, I repeat, Foster was a supreme judge of men.

When Gaskell determined to take up physiology as a career, Foster advised him to go to Germany and study under Ludwig, who was then introducing mechanical methods of investigating physiological problems. Particularly was he studying the cardiovascular system with the aid of his kymograph. It is not surprising therefore that on his return to Cambridge a year later in 1875 Gaskell started his independent work by researching on that system and the relationship of the sympathetic nervous system to it, for from the first this was his chief interest. He began by studying the influence of chemical substances on the calibre of the peripheral blood-vessels. Finding that the muscles of the arterioles were responsive to other than nervous stimuli, his interest in the responses of heart muscle was aroused by some observations that had just been made by Foster and Dew-Smith on the automatic rhythm of the invertebrate heart. This was one of Foster's rare

incursions into experimental research. His collaborator Dew-Smith was a remarkable man. He had returned from the South Seas where he is credibly reputed to have been the original of Attwater, that strange character in R. L. Stevenson's and Lloyd Osborne's *Ebb Tide*. He was one of the founders of the Cambridge Scientific Instrument Company, and the first of artistic portrait photographers. Indeed his photograph of Michael Foster is much more lifelike and characteristic than the Herkomer portrait.

Gaskell already impressed with the fact that nerve stimuli were not the only potent ones, devised a method to determine if automatic rhythm could also be demonstrated in the vertebrate heart. Up to that time its rhythm had been attributed to the nerve ganglia it contained. Bernstein had shown that if the ventricle of the frog's heart were "physiologically disconnected" by crushing the

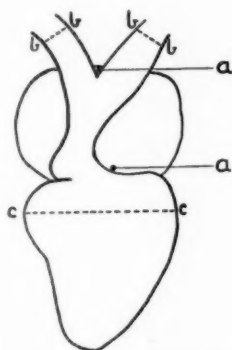


FIG. 2.—Diagram of frog's heart showing position of (a) nerve ganglia; (bb) site of ligature of aortæ, (cc) site of "physiological disconnection".

auriculo-ventricular junction with a fine pair of wire forceps, it remained quiescent, while the rest, which contained ganglion cells, continued to beat. But Gaskell simply raised the intracardiac pressure by ligaturing the aortæ, and saw the ventricle begin to beat rhythmically once more. To my mind at that moment and by that experiment modern cardiology was born, although more than twenty years were to elapse before the clinical applications were made.

In the tortoise's heart he was able to divide the septal nerve which passes between the two intracardiac ganglia without disturbing the rhythm; and by a series of interdigitating cuts in the auricular substance he compelled the wave of contraction to pass along a zigzag strip of muscle between the sinus venosus and ventricle, though all nerves must have been divided. Finally, by warming the ventricle and cooling the sinus, he was able to alter the relative excitability of the two ends of the heart so much that a reversed rhythm was produced. His conclusion was that rhythm was an inherent property of the cardiac muscle, and did not depend on the intracardiac ganglia. The beat normally began at the sinus, because here the muscle was of a more embryonic character, while the ventricular muscle was the most differentiated.

He went on to show a point which has now become of great practical importance. If the bridge of auricular muscle be made too narrow by cutting, a "block" is established on the course of the muscle wave, so that not every beat can pass over into the ventricle, but only alternate waves, or one out of every three, according to the width of the bridge. But after inhibiting the heart by stimulation of the

vagus, the muscle accumulates enough energy during the enforced rest to enable it to convey every beat across the narrow bridge. On the other hand, in the period of comparative exhaustion following sympathetic stimulation, the conductivity is lowered, so that fewer beats can pass over. Here was the first suggestion of an idea which has been fruitful, namely that the parasympathetic is preponderantly anabolic, and the sympathetic katabolic in its activities. An adequate strand of conducting tissue is essential to the due propagation of the wave of contraction along the cardiac tube.

Before these results could be applied to the mammalian heart it was necessary to prove the existence of muscular continuity between auricle and ventricle, which at that time was not thought to be present. Stanley Kent was the first to do this, in 1893; but His, and then Tawara, worked out the nature of the connecting band or "auriculo-ventricular bundle" in much greater detail. The branches and terminal filaments of the bundle resemble those fibres which were described as long ago as 1845 by Purkinje. Morphologically and histologically these fibres represent the invaginated portion of the primitive tube from which the complex heart of the mammal is built up.

Later Keith and Flack found another remnant of primitive fibres persisting at the sino-auricular junction. Here the dominating rhythm of the heart normally arises and here it may readily be modified by extrinsic nerves.

The anatomical evidence is therefore amply in favour of extending Gaskell's conceptions to the mammalian heart. And Hering was able to produce a complete stoppage of the supra-ventricular parts of the heart by a cut made at the sino-auricular junction; while Erlanger has shown experimentally that it is possible, by interfering with the auriculo-ventricular bundle, to reproduce the phenomena described by Gaskell in the tortoise's heart, and many of the forms of irregularity met with clinically, particularly heartblock.

The clinical applications of all this were initiated, as is well known, by James Mackenzie. His approach was purely clinical but he himself once said to me, "the further I go, the more I realize that Gaskell was the man". For in Gaskell's pioneer work he had found the explanation of many of his own observations on disturbed heart rhythm. Einthoven's string galvanometer proved to be the key which opened the door to further discoveries, and by its aid, particularly in the hands of Sir Thomas Lewis, modern cardiology has been built up. It is of great interest to find in Lewis's later work on the peripheral circulation a confirmation of the idea in Gaskell's first paper that it might be profoundly modified by chemical agents.

While insisting on the automatic nature of cardiac rhythm, Gaskell naturally did not disregard the influence of extrinsic nerves upon it. Rather did he clear up some outstanding difficulties in understanding that influence. For it had puzzled observers to find that the effect on the frog's heart of stimulating the vagus was variable. Gaskell showed that in the frog, the tortoise, and crocodile, this nerve was really compound, a vago-sympathetic trunk, the two latter animals being especially suitable for his purpose as the intracranial vagus in them is long and easily stimulated. If he stimulated the sympathetic before it joined the vagus, the effect was always acceleration, while if he stimulated the intracranial vagus before the sympathetic had joined it, the result was always inhibition. This antagonism between their functions led him on quite logically to study the plan of the sympathetic, comparing it and contrasting it with what we now call the parasympathetic nervous system.

Here we enter on the second phase of Gaskell's great work. I have often said that to read an account of this system before Gaskell is like reading an account of the circulation before Harvey. His part in its elucidation has sometimes been belittled, and it has been pointed out that in his later descriptions he incorporated much of the work of others, particularly of Langley's. That is perfectly true and it was only reasonable to do so, since he gave credit for such other observations where it was

due. In order to be quite clear on this matter I have gone back and read again Gaskell's original papers published before other researchers had taken up the tale. From this you will see how broad and comprehensive was his vision. Like all great discoveries, the basis was quite simple. He noted that the voluntary nerve fibres were of a larger calibre than the involuntary ones.

His first great paper on the subject appeared in 1886 in Volume VII of the *Journal of Physiology*, where he boldly enunciated the generalization that the small calibre

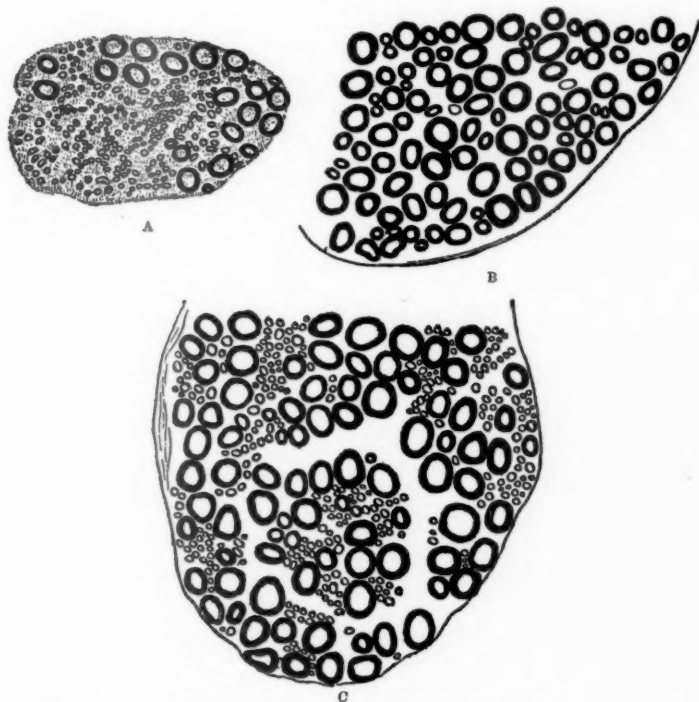


FIG. 3.—Sections across parts of the roots of various nerves of the dog, to show the variations in size of their constituent fibres (Gaskell). A, from one of the upper roots of the spinal accessory. B, from the first cervical anterior root. C, from the second thoracic anterior root. The small calibre of the fibres with visceral functions is clearly shown in A and C. Such fibres are absent in B.

(From "The Sympathetic Nervous System in Disease", by W. Langdon-Brown. With acknowledgments to Messrs. Hodder and Stoughton, Ltd.)

of a medullated nerve might be taken as evidence of its visceral function. Such visceral nerves issued from the central nervous system in three definite groups—cervico-cranial, thoracic and sacral, separated from each other by the great limb plexuses. The second of these was the sole source of the outflow of the sympathetic nerve fibres, which left the spinal roots by the white rami communicantes—the grey rami being recurrent fibres which having lost their medulla in a ganglion were being redistributed along spinal nerves. All vasomotor nerves left the cord by the white rami. You will note that he uses the word vasomotor where we should now say

vasoconstrictor; this was because he regarded vasodilators as inhibitors, equivalent to the cardio-inhibitory fibres. He showed that the visceroinhibitory nerves were also sympathetic in origin, while the visceromotor and the vasoconstrictor had their outflow in the cranial and sacral visceral fibres. By proving that the grey rami had no direct communication with the spinal cord he revolutionized the view previously held by Bichat and others that the sympathetic was an independent system which reciprocally interchanged fibres with the spinal cord.

You will see that at this early stage of his research he had already clearly grasped the essential points; the breaking up of the visceral outflow into three main sections, separated by the great limb plexuses where the nerve roots were entirely somatic in type; the antagonism between the functions of the sympathetic and what we now call the parasympathetic; the curious balancing of visceroinhibitory and vasoconstrictors in the former against the visceromotor and vasodilators in the latter. But even in this first paper he had gone on to consider spinal segmentation in terms of somatic and splanchnic roots, and had indicated which groups of cells in the cord were the probable origin of the visceral fibres. He went further, for having noted the shift in segmentation of the visceral outflow which had resulted from the interposition of the limb plexuses, he applied a similar type of reasoning to unravelling the segmentation of the brain. It is unnecessary here to go into his conclusions as to the rearrangement which had occurred in the brain segments, but it is important to mention the fact as this was one of the considerations which led him on to his theory of the origin of the vertebrates.

I think you will agree that in this paper the main plan of the visceral or involuntary nervous system was clearly laid down. As questions of priority have been raised I may quote Langley's statement of the position before this paper appeared. He said "Those writers who tried to give an impartial summary of the state of knowledge found themselves reduced to stating a number of more or less contradictory facts and irreconcilable theories".

Gaskell called attention to the arrangement of the lateral and collateral sympathetic ganglia, but was under the impression that one of their functions was to effect the conversion of medullated into non-medullated fibres. This we now know to be incorrect, but it is only right to remember that at that time the histological methods of Golgi and of Ramón y Cajal had not established the entity of the neuron as a unit, contiguous to but not continuous with neighbouring units. The discovery by Langley and Dickinson in 1889 of the paralysing effect of nicotine on the fibres entering the sympathetic ganglia while not affecting the fibres that left them, put into their hands an extraordinarily effective method of tracing out the ultimate distribution of the sympathetic. This led moreover to the generalization that every impulse in the autonomic system passed through two and only two neurons, the first starting from a cell in the lateral horn as a fine medullated pre-ganglionic fibre and ending round a ganglion cell, whence the non-medullated post-ganglionic fibre proceeded to its destination. Whereas the sympathetic nerves make their cell connexion early, the parasympathetic do not do so until the peripheral, terminal ganglia are reached. The nicotine method enabled Langley to reach a very accurate conclusion as to the cutaneous distribution of sympathetic fibres as evidenced by the pilomotor and sweat nerves.

I have thought it worth while to labour these points, because it speedily led to the first clinical application of Gaskell's work—namely the interpretation of referred pain, by Ross, James Mackenzie, and Henry Head successively. Yet the real clinical significance of the rest remained unappreciated until W. B. Cannon published his book on *Bodily Changes in Pain, Hunger, Fear and Rage* in 1915, a time lag even greater than that in the clinical application of his work on the heart.

Already I have mentioned that Gaskell's work on the segmentation of the brain and spinal cord led him on to the subject to which he devoted the remainder of his

life—the origin of the vertebrates, a subject which became acutely controversial. To appreciate the position we must realize that there was at that time a very active school of animal morphology in Cambridge. Alfred Newton was the Professor of Zoology, but his interest was almost entirely confined to birds. Frank Balfour, the brilliant brother of Arthur Balfour, initiated the study of embryology, and based his views of morphology on the recapitulation theory, according to which, as it was popularly expressed, every animal climbed up its own genealogical tree; a view less in favour to-day, but one which seems to me very helpful. He was gathering round him a group of collaborators, scarcely less brilliant than those under Foster, when he met his death climbing in the Alps in 1882. But his influence continued, and although it is practically certain that such an active mind would have gone on growing and changing its outlook, it came to be almost heresy to differ from the views he held at the moment of his death. Yet we realize now that not only may form influence function but that function may fashion form. Gaskell approaching the subject as a physiologist naturally attached more importance to function than the morphologists. There was and, I think I may add despite Gaskell's fascinating contribution to the problem, there still is no entirely satisfactory theory to account for the origin of the most dominant group of animals in the world—the vertebrates. The morphologists were searching for it among such weirdly aberrant forms as *Amphioxus*, *Balanoglossus* and the *Tunicates*. Gaskell laid stress on the orderly sequence in the development of the central nervous system, the master structure in which no break of continuity can possibly have occurred. He coined the aphorism "The race is not to the swift or to the strong, but to the wise"—"the secret of evolutionary success" he said "is the development of a superior brain". Amid all the dissolution of tissues which occurs in the chrysalis stage between caterpillar and butterfly the nervous system remains intact. In the transformation of the larval *Ammocoetes* into the adult Lamprey on the other hand, a new alimentary tract is formed. Now in the invertebrates the ganglia which constitute the brain surround the gullet. Thus in evolution they encountered a terrible dilemma; if they developed their brains they would not be able to swallow their food; if they did not, they were debarred from further advance. Indeed the most intelligent invertebrates are compelled to live by sucking blood from other animals. The evolution of the vertebrate type provided a way of escape from this dilemma, for there the brain no longer encircles the mouth. Gaskell's starting point was the recognition of the close similarity in structure and function of the different parts of the vertebrate brain with those of arthropods as Gegenbaur had already indicated. He regarded the vertebrate central nervous system as consisting of the fusion of two originally independent structures, one nervous, segmentally arranged on the arthropod plan, which surrounded the other, a tube lined with epithelium, now the ventricles of the brain and central canal of the cord, but representing the remains of the arthropod alimentary tract. That would amply explain Rathke's pouch as the stomodaeum of the old alimentary tract, with the pituitary representing the green gland of the arthropod and at the other end, the neurenteric canal. On any other theory it is difficult to explain the reason for a fetal communication between the central nervous system and the existing proctodaeum.

One morphologist, Dr. Hans Gadow, appreciated the cogency of Gaskell's reasoning. He wrote:

"This idea explained at once the remarkable non-nervous epithelial parts of the tube, which become so conspicuous as we descend the vertebrate phylum, and every part of this tube bears the same resemblance to various parts of the C.N.S. as the dorsal stomach and intestine of an Arthropod. As a crowning of his conception the pineal eyes fit into the right place of the scheme; and the resemblances become greater and more numerous on the one hand in *Ammocoetes*, as was to be expected in the lowest available vertebrate, and on the other in *Limulus*, the King crab. In short, there was now a provisional working hypothesis, obtained by a direct logical process from the consideration of the vertebrate nervous system."

Other morphologists, however, seemed to resent the incursion of a physiologist into their domain. The discussion which followed the reading of his paper before the Cambridge Philosophical Society became somewhat heated. Gaskell humorously replied that whereas he considered the brain the most important organ, his chief opponent apparently considered the stomach was!

When Le Verrier and Adams were able from the perturbations in the orbit of Uranus to predict the discovery of a new planet and to fix at a given moment its exact position in the heavens, it was rightly regarded as a singular triumph for Newton's theories. Such predictions are valuable confirmation of the theory which enables them to be made. By this theory Gaskell was enabled to predict the discovery of so many anatomical and physiological facts that he gained new support for his views. Perhaps one of the most unexpected outcomes of his theory is the better comprehension it provides of the biology of the endocrine system, the very existence of which was hardly appreciated when the theory was first formulated. That is the reason for my discussing his theory here. I have attempted to detail the evidence elsewhere (*New York Med. Journ.*, April 5, 1922) and will only summarize it now. Endocrine glands appear to be modified nephridia. To this rule the only exceptions are the medulla of the adrenals and the cell islets of the pancreas. In the worms and a lowly arthropod such as *Peripatus*, paired nephridia open externally in each segment. With the development of appendages these undergo modification in the different regions of the body, the most anterior becoming the pituitary, the middle group giving rise to the tonsils, thyroid, parathyroids, and thymus, the third forming the cortex of the adrenal. Only the hindmost retained their original excretory function, becoming conglomerated into the metanephros, and draining into the metanephric duct instead of opening separately to the surface. The others were forced to become ductless by the formation of the new alimentary tract and pleural folds which shut them off from the surface. But just as the nephridia maintained the constancy of the internal medium of exchange, so their successors contribute to that process by internal secretion instead of external excretion. The endocrine functions which are the specialization of the old chemical methods of stimulation and defence became concentrated in these structures which perforce had to lose their excretory functions. We are reminded of the hermit crab that seizes on an empty whelk shell. And just as the hermit crab existed before it found an empty house, so the endocrine functions were in existence in a less specialized form before they had a local habitation. Gradually these chemical methods came under the control of the sympathetic nervous system to a considerable extent, which correlates them and enables them to be brought rapidly into action either for the ordinary processes of metabolism or for external and internal defence.

I have failed in my purpose if I have not made clear the very logical sequence of Gaskell's ideas. A mathematician turned physiologist, he was naturally first attracted to exact methods of recording physiological observations. Applying these to the changing calibre of arteries he found that nervous stimuli were not the only effective agents. This led him to investigate the rhythm of the heart and to show that it too was not initiated by intracardiac nervous structures, but by some intrinsic metabolic factor. At the same time he recognized that the extrinsic nerves had a profound influence upon it, and by studying the antagonistic effects of the vagus and sympathetic in this, went on to discover the distribution of such antagonistic effects throughout the visceral nervous system. This resulted in an entirely new anatomical conception of the sympathetic and parasympathetic, and thus to a new view of the segmental arrangement in the brain and cord. Comparing this segmentation in vertebrates and invertebrates he thought that he had a solution of the problem of the origin of the vertebrates. Whether that solution is accepted or not he has, as Gadow said "discovered and elucidated many a feature both in

vertebrates and invertebrates which without his tireless work would remain still neglected and unexplained". It is his contribution towards the elucidation of the origin of the endocrine system to which I would direct your attention as of topical clinical interest.

Indeed it is of special interest in view of recent biochemical advances that Gaskell throughout should have firmly believed in the interaction of nervous and chemical mechanisms. We see it in the first paper he published wherein is the idea of metabolites influencing the peripheral circulation; we see it in his experiments on the effect of muscarin and other drugs on the cardiac vagus which foreshadow many of the later observations detailed in Professor Fraser's Croonian Lectures for 1938: we see it finally in his conception of internal secretory functions based on nephridia. His son Dr. J. F. Gaskell in a private communication to me says:—

"He always held that there was an internal secretion adjuvant to nerve action such as was proved by Elliott and others in the case of adrenalin and the sympathetic, and he was much excited about Dale's work showing the relation between the action of the parasympathetic and acetyl-choline. He prophesied that similar adjuvant secretions were probably present for all muscular systems."

Here again he was a true prophet.

In summing up the influence of all this may we not say that it has largely contributed to that new conception of psychosomatic unity which is now taking shape before our eyes. As I see it, it is something like this. Centres for emotional expression in the basal ganglia can receive impulses both from the external world and the grey matter of the cortex. They may in consequence initiate activities through the hypothalamus, either by way of the sympathetic of which it is now regarded as the head ganglion, or by way of the pituitary, which may be regarded as the transformer of nervous impulses into chemical energy, through which it can influence the other endocrine glands. And this is only a specialized instance of such a transformation, now that thanks to the observations of Loewi and of Dale we know that all nerve terminals are cholinergic or adrenergic. Thus as Hopkins phrased it, chemical substances translate for the tissues the messages received from the nerves. While the endocrine system carries on an autonomous life of its own, as does the heart, like the heart it can be played upon by these direct and indirect stimuli, according to the needs of the organism. Thus emotional states may produce visceral effects, and a functional nervous disorder lead to physical changes. The sympathetic nervous system is designed to work as a whole but in disease its functions may be dissociated and there may be perseveration of its action until structural changes may result. Perhaps too much stress has been laid on the influence of the endocrines on behaviour, without realizing how much the emotions may influence their secretion.

The medicine of the twentieth century has been much interested in a new cardiology, in the rôle of the autonomic system in disease and in endocrinology, together with the interactions of these three. It is an astonishing thing that one man, and he not a clinician, should have laid down the entire foundations of two of these, and have thrown light on the plan of the third. The debt of modern medicine to Gaskell is enormous. This is not to belittle the achievements of others. The splendid accuracy of Langley's mental microscope extended and completed the discoveries made by the great sweep of Gaskell's mental telescope.

It is a curious and interesting fact that two such different characters as Gaskell and Langley, starting from such different standpoints should have reached convergent and confirmatory results. Gaskell was essentially a big man, alike in physique, personality and character. He poured out ideas with unstinted prodigality, often giving them to his pupils to develop and leaving them to take the credit. He delighted in sweeping generalizations in which, though they were

sometimes based on inadequate detail, he was guided aright by his philosophic insight. To attend his lectures was an exciting experience, for as Langley said: "his style was incisive, and he spoke on controversial points with a half-suppressed enthusiasm which was eminently infectious". I should like to add to that tribute how much we appreciated his little personal chats, sitting by us at the laboratory benches. He made us feel that we were fellow explorers with him in a fascinating adventure. Sir Henry Head wrote of him in the *Dictionary of National Biography*,

"He was ever ready to turn aside to give counsel and encouragement. . . . Those who came under his influence can never forget his transcendent sincerity, his gift of sympathetic attention and the unflinching wisdom of his advice."

In these hurried days there is something to be gained from contemplating his leisurely way both in work and recreation. His methods were certainly justified by the results. For the last twenty-two years of his life he dwelt on a hilltop in Great Shelford, opposite that on which stood Michael Foster's house. Here he cultivated his garden with its charming terraces. Here, having just revised the last sheets of his book on the *Involuntary Nervous System*, he died suddenly in his 67th year on September 7, 1914, secure in the affectionate remembrance of all his old students. For to have known him was an inspiration.

I can hardly hope to have been able to make the history of those days as interesting to you as I have found them in the retrospect. The men concerned stand out as vividly in my memory as though it were a story of only yesterday, for they have coloured so much of my subsequent clinical interests. It was admittedly a great epoch in British physiology, and I am thankful that I had the opportunity of living in it.

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## Section of Orthopædics

President—T. P. McMURRAY, M.Ch.

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### The Organization of Hospital Services for Casualties due to the Bombing of Cities, Based on Experience Gained in Barcelona— —with Special Reference to the Classification of Casualties

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ABSTRACT.—(1) Difference between modern "total population" war and old-fashioned war. Difference between bombing of (a) military objectives and (b) civilian population.

(a) The heavy bomb, e.g. 750 lb., with large fragments, upward throw, great destruction of buildings.

(b) The light bomb with finger nail fragments, horizontal throw, great velocity.

There is in addition the incendiary bomb, little used in Barcelona because the buildings are built of stone and concrete.

(2) Aerial bombing of a town produces injuries needing more immediate hospitalization than most front-line wounds. At the same time it is possible in a town to organize rapid collection of patients and their immediate transfer to hospital.

(3) Experience shows that it is most desirable to make this transfer of patients to hospital a primary consideration. On arrival they are "sorted" and minor injuries are given *First Aid* treatment and sent home, others are fully examined, *classified*, and dispatched to the theatres on a *priority* list, to nearby wards for resuscitation, to wards for rest, or sent on to plaster rooms for splintage, or to a neurosurgical centre.

(4) First-aid posts in a town should be in hospitals and treat superficial injuries, &c., after primary sorting in the hospital reception room.

(5) First-aid posts in outlying areas should carry out the same function for the same type of cases; all the more seriously wounded, including those with tiny penetrating wounds, should be dispatched without first aid treatment direct to hospital.

(6) *Hospital arrangements*, for circulation of ambulances, for sorting, undressing of patients, docketing of valuables, &c.

(7) *Classification* must be carried out by surgeons of experience and judgment. They must regard not only a standard priority list but the particular clinical picture and prognosis in each case.

(8) The surgeon will furthermore draft the cases with regard to the special abilities of the surgical units available, e.g. chest, abdomen, or limbs.

(9) Review of wounds in limbs, chest, abdomen, and head, caused by fragments from heavy bombs or by splinters from small light bombs.

(10) Injuries from falling masonry.

(11) Standard classification by urgency of operation.

(12) Classification re possible early evacuation.

THE characteristics of modern war are different from those found in all the former ones. The Spanish struggle, especially in Catalonia, is the first in which the new tactics have been employed. My personal experience during the aerial bombardment of Barcelona and several other towns and villages, chiefly those of Granollers, Mollet, Badalona and Figueras (covering altogether more than three hundred and forty air raids) permits me to give you a review of the most important points which differentiate war in open towns from that in the front line.

With the new conception of what may be called the "total population" war, attacks against open towns can be employed with two objects:—

Firstly, against military objectives.

Secondly, against the civilian population.

In the first type of aerial bombardment the material used in attack is different from that in the second. The bombs usually employed are heavy ones, more than six hundred pounds each, and the explosion produces very grave damage to buildings. I have seen several buildings strongly constructed in stone or concrete, eight floors high, completely destroyed by bombs of three hundred kilos weight (about seven hundred and fifty pounds). When these bombs fall in the streets they produce a hole about ten feet deep, and there is an enormous dispersion of shell fragments in an upward direction.

The wounds caused by these bombs are very severe and very few cases injured in the chest, head or abdomen survive. Transport of all these casualties is very difficult and shock and hæmorrhage increase with the movement. Immediate evacuation to the nearest hospital followed by complete treatment is the only thing—which can save some of these wounded.

The second type of casualty produced by these heavy bombs includes those injured by the collapse of the buildings as a result of explosions. These, too, are often in a grave condition, with the exception of some who are fortunately protected by means of some big stone or beam.

All these casualties (those produced in the streets, and those injured in the buildings) need immediate assistance and even with this, the mortality is very high. Without the best organization of brigades for extricating victims from the debris of fallen buildings and means of immediate transport to hospital where most efficient treatment can be given, the mortality statistics become tragic.

Another type of bomb can be employed against the buildings, viz. the incendiary bomb which is small and weighs two or three pounds so that several hundreds can be carried by each aeroplane. My personal experience of incendiary bombs is small, as the buildings in Catalonia, and particularly in Barcelona, are composed mainly of stone, steel and concrete, and when the enemy found that the damage done by these bombs was unimportant their use was abandoned. On the other hand in those towns in which wood entered largely into the construction of the buildings, such as Madrid and especially Guernica, incendiary bombs were employed frequently. From the descriptions of the aerial attacks on Warsaw it appears that incendiary bombs were used considerably in the Polish campaign. Most of the fires that occurred in bombardments of Barcelona, resulting in severe burns among the casualties, were produced by explosions of gas in buildings damaged by heavy bombs.

## AERIAL BOMBARDMENT OF THE CIVILIAN POPULATION

The second type of aerial bombardment is that made by light bombs, against the civilian population in the streets, for the purpose of producing terror. These bombs are covered with a light material like aluminium, and produce thousands of little splinters, many of them no larger than a finger nail. The light bombs produce very shallow craters in the ground. When they explode, the dispersion of the splinters is horizontal, and for that reason the largest number of wounds are in the leg or in the lower abdomen. Generally these people can be moved longer distances but the deep damage in the muscles is great and is out of all proportion to the small wounds produced in the skin. Many of those injured suffer from shock, and serious hæmorrhages occur. First Aid outside the hospital is useless, for they *must* be treated by operation in a hospital within two hours. The fact that all these people are wounded in the street makes it easy to reach the hospital within this time.

## DIFFERENCE BETWEEN BOMBED TOWNS AND THE FRONT LINE

As you can see by this description most of the casualties in a bombed town are grave and need treatment as quickly as possible. On the other hand, in the front line many casualties are produced by rifle and machine-gun bullets, and also by shells which, if they do not explode very near, are relatively not so destructive as aerial bombs, thus partly compensating for the delay in reaching some place where skilled surgical treatment is available.

The constantly changing position of the front line, even in trench warfare, needs the establishment of hospitals at a sufficient distance behind the lines, so as to give time for evacuation in case of retreat, and always out of reach of the heavy artillery.

In the front line the collection of the fallen continues systematically, each man being taken as soon as possible after being wounded, and the *minor first aid* in an advanced post (for dressing, to place a splint or a tourniquet) is always necessary. Military organization with strict discipline, the responsibility of the officers, and the absence of women and children, permit the maintenance of good morale.

In open bombed towns, the general gravity of the casualties, the proximity of the hospitals, the production simultaneously of all the victims, the number of the women and children wounded by the bombs, the presence of the victims' families in the same place, and the absence of a rigid discipline, compel us to organize assistance for the victims under different schemes from the military ones. I think this new method of war has more in common with towns besieged by armies than with fighting in the front line, as was suggested in an interesting letter published in the *Lancet* of May 6, 1939. Similar problems were presented in besieged towns, when artillery fire killed or wounded the civilian population and destroyed the buildings; here too the hospitals like other structures were in danger.

Many surgeons who have experienced the difficulties of carrying on surgical work in besieged towns would object that in bombed towns there is the possibility of carrying away victims to safe hospitals as soon as they are wounded, but I can assure you that with air raids there is no safe place, but perhaps the most secure are those properly defended by modern equipment such as anti-aircraft guns, balloon barrages and fighter planes. This protection is confined to the more important towns; in the country or in the little villages it is easier to destroy the objectives. My experience proves this, as you can see from the following examples:—

## HOSPITALS WITHIN THE DEFENDED AREA OF THE TOWN.

Hospital Clinic de Barcelona..	..	..	Never hit.	
Hospital General de Catalunya	..	..	Never hit.	
Hospital de la Creu Roja	..	..	Never hit.	
Hospital Cardenal	..	..	Never hit.	Three bombs fell close to it.
Hospital de l'Aliança	..	..	Never hit.	

## HOSPITALS OUTSIDE THE DEFENDED AREA OF THE TOWN.

Hospital de Reus	..	..	..	Completely out of action.
Hospital de Caspe	..	..	..	Hit several times.
Hospital de Figueras	..	..	..	Completely out of action. In one raid alone, 83 killed and 118 wounded.
Hospital de Flix	..	..	..	Completely destroyed.

The reason for this difference between the destruction of the hospitals in the defended area and outside it, depends on the lack of precision which occurs, since the attacking planes must fly at great altitudes. The attacks on the defended area of Barcelona were effected from a height of *more than sixteen thousand feet*; those on undefended villages were effected from *less than five thousand feet*. The attacking planes may not aim specifically at hospitals, but the relative size of the hospital buildings in a small congested town makes them particularly vulnerable. You can see the lack of precision in the defended areas in the following data :—

- (a) The electric power station, situated in a street of Barcelona, was almost constantly attacked but was only once slightly damaged.
- (b) The artificial silk factory at Blanes, a village situated forty miles from Barcelona, was attacked several times. In the first raid it was rendered useless, and in the second it was entirely destroyed.

Road and rail communications between the town and outlying hospitals are liable to be cut by air attack, especially if bridges intervene, and ambulances may be attacked *en route*. Experience in Barcelona proved this. During the last period of the war in Catalonia, patients being moved from the Hospital de la Savinosa and Hospital de Sitges were constantly attacked *en route* by enemy planes and many casualties occurred.

*First Aid Posts.*—For all these reasons it is best to organize the First Aid Post in the hospitals of the bombed town, since more than 30% of patients need operation. Whatever tends to facilitate the rapid admission to hospital of air raid casualties contributes to the more favourable outcome of the treatment, and thus it is essential that there should be no hindrance whatever to the immediate admission of casualties. Sometimes hindrance is due to defects in the organization itself, as happened in Barcelona when "emergency treatment posts" were set up inside ambulances, or in the so-called "First Aid Post" organized in different parts of the city without connexion with the hospitals.

The impossibility of foreseeing the intensity of the aerial bombardment, the nature of the bombs to be used, the time of the attack, and which districts will suffer, prevent one from having ready all the resources which would make successful treatment certain. On the other hand there are important factors of *morale* which succeed in disturbing and diminishing the efficiency of the major measures relied on. I refer to the acute agitation which takes possession of many highly strung people and which can be communicated to the surgical centres in heavy bombing raids especially during the first attacks when the people are not accustomed to them. The standard of efficiency is in inverse proportion to the extent of fatigue of the personnel of the hospitals after the heavy bombardments, especially that of the surgeons, as for

example, in the bombing of Barcelona which persisted for three days in the month of March 1938. There we had, in our hospital alone, more than 60 doctors and we worked without interruption for nearly three days; the surgical units being completely exhausted after performing 731 operations.

For these reasons the best possible organization is necessary, and I wish to speak about the most important points which arise, after the experience I gained during thirty months of air attack.

Speed of transport is essential for successful treatment; in Barcelona I operated on a victim who had been brought about 5 miles within eighteen minutes of his being hit. That was, of course, exceptional, but generally about twenty to thirty minutes must be allowed for a distance of 5 miles. This dispenses with so-called first aid, and after the tragic experience of Barcelona I maintain that the *immediate intervention of the surgeon* in hospital is the only useful form of first aid other than the application of a tourniquet to the limb which bleeds excessively, and this can be applied by the ambulance man. The application of a splint, bandage or other form of first aid has been shown in my city not only to be useless, but actually harmful, because it always delays the surgical treatment.

*Evacuation.*—I understand that in many cases it will be difficult to keep the victims in the city hospitals, because of the lack of beds. For this reason it is best to evacuate from town as many of the casualties as possible after they have received surgical treatment; 70% of cases can usually be moved. Under these conditions the evacuation is easy, the time lost in travelling is not so important because the only thing which the treated patient needs is rest and all the evacuations can be made without haste and without the accumulation of patients in the beginning of the bombardment. Trains are more suitable than ambulances for long travelling. A great number of these evacuations, especially of those operated on, can be made during the night, and the victims can be sent far from the range of the attacking planes.

First Aid Posts, such as were organized in Barcelona during the first phase, can be useful in connexion with the hospitals, or for reassuring nervous people. A first aid post in a hospital can be used for the treatment of the slightly wounded, but only *after* they have been examined in the classification room. As I will describe later, the type of wound depends on the kind of bombardment, but it is very common to employ different types of bombs at the same time. I have seen few bombardments in which only heavy bombs were used. In most cases both heavy and light bombs were employed but where the object of the attack was to produce terror and demoralization among the civil population light bombs only were used.

In most bombing raids there are people injured by falling masonry, others wounded by big fragments of heavy bombs which have fallen in the streets or by tiny high velocity splinters from light bombs. In some raids, especially in the undefended areas, it is common to see people wounded by bullets fired from the planes by the machine-guns. I have seen many such casualties sent in from outlying small towns but they never occurred in Barcelona because the anti-aircraft defence forced the attackers to fly high so that they could not employ these tactics.

The casualties which arrived first at the hospital were those wounded in the streets, and generally they are less seriously wounded than those in the fallen buildings. Unfortunately the latter, whose condition is more dangerous, reach the hospitals when everybody is busy with the first arrivals.

*Hospital arrangements.*—For the smooth running of the work in the hospitals, perfect organization is necessary outside for the circulation of ambulances, but it

is even more vital inside the hospitals themselves. The latter point, which at first sight would seem to be of little importance, can cause a great deal of confusion if not properly solved. We may take as an example the difference in efficiency between the two largest hospitals in Barcelona: the Hospital Clinic with 1,600 beds and the Hospital General with nearly 3,000 beds. In the former, the problem of one-way traffic for the ambulances and their rapid unloading had not been solved; on several occasions they were found to be in utter confusion. In the Hospital General because of its special shape and of the existence of large gardens between the buildings, one-way traffic facilitated rapid unloading of the ambulances as they arrived.

The crucial point for the efficient running of the work inside the hospital depends on the existence of perfect co-operation between all the services, especially between the centre for the classification of the wounded and the separate operating theatres, wards and exits of the hospital so that the casualties can be distributed into five groups:—

(a) Those who required immediate operation, and whom it is impossible to send out of the hospital after treatment.

(b) Those who require immediate operation and who can be evacuated afterwards.

(c) Those who need immediate treatment and rest without operation and cannot be evacuated.

(d) Those who after receiving first aid can be transferred to the base hospital for further treatment.

(e) Those who can go home after immediate treatment.

It is very important to have a great number of small record cards or labels in five colours; in the classification room each injured person is given the appropriate card. This precaution avoids many mistakes when the bombing is heavy.

If the classification unit functions under good technical direction, and there are a sufficient number of assistants available immediately to sort out all the cases which arrive at the same time, the work in the other sections of the hospital is greatly reduced.

The director of the classification unit should know the number of surgeons and operating tables available and the speciality of each surgeon, so that the cases are distributed according to the capacity of each one, and in relation to the fatigue of the operator.

In every heavy bombing raid, the number of available surgeons proved to be small in relation to the needs. We saw ourselves forced to include in the surgical units general practitioners and specialists in the capacity of assistants. In the Hospital General de Catalunya, we established shifts of twenty-four hours, with six units, each made up of eight doctors. One of the units remained in the hospital for twenty-four hours consecutively and the others were prepared to intervene if necessary, the doctors holding themselves in readiness in their homes. In every team there must be some specialists, including always ophthalmologists, because many of the wounded have eye injuries associated perhaps with several wounds in other parts of the body.

#### CLINICAL CLASSIFICATION OF WOUNDS

In many raids there are casualties so seriously wounded with multiple fractures, internal hæmorrhage or crush injuries of the chest or abdomen, that no surgical treatment is of any use.

Features common to all those injured in air raids, especially those who have been wounded by falling buildings, are the great fall of blood-pressure, the pale

colour of the face, and the clear consciousness; occasionally there is mental excitement, but mental depression is rare except in those almost moribund. It is very impressive to see a great collection of wounded directly after an air raid because of their extreme stillness and the absence of crying or moaning in spite of serious wounds, which contrasts with the excitement and noise made by friends and relations.

The following table shows the distribution of injuries in different parts of the body, analysed from a total of 9,850 patients treated in different hospitals in Barcelona. It does not include many who died before admission to hospital; these would raise considerably the cases of multiple injury.

Multiple major injuries .. ..	32.6%
Lower limb, including hip .. ..	22.3%
Upper limb, including shoulder ..	14.7%
Thorax .. ..	12.1%
Abdomen .. ..	9.1%
Head (skull and face) .. ..	8.2%

Many of these patients had minor injuries in other parts.

The types of injury produced by the bombs can be classified into two groups:—

(1) Wounds produced by big shell fragments coming from heavy bombs directed against the buildings but which fall on the streets. These bombs cause great damage to the tissues; the wounds resemble those produced by artillery fire but are often more bruised, probably because the speed of the fragments is greater.

*In the limbs* it is common to see those injured by such shells arrive in hospital having lost one or more extremities, with great trauma to the tissues, especially the muscles. In amputating it is always necessary to remember the retraction of the bruised muscles. In several cases I have seen infective complications because of insufficient operative exposure, especially when the incision was not carried high enough, thus leaving devitalized tissue which is a favourable site for the development of gas gangrene.

The lesions of the arteries are always irreparable; and primary suture of the nerves is contra-indicated in those rare cases in which it is practicable.

*In the abdomen the wounds* produced by these bombs are always most grave. Many of these patients are dead on arrival, and others are brought to the hospital with great eviscerations which make attempts at intestinal reconstruction impossible.

Among the large number of such cases that I have seen, I can only remember two that survived, one of whom was an engineer with a penetrating wound in the lumbar region, which entered the large intestine (descending colon) and who was operated on twenty-three minutes after being injured.

*In the thorax* large fragments of shell produce "open chest wounds" which are dangerous because of hæmorrhage and pneumothorax. The surgical technique in these cases is very difficult, as the patients are greatly shocked and it is necessary in many cases to perform partial pneumonectomies. Thoracic reconstruction is usually difficult because of the loss of tissue. When the first danger is over it is very common to see infective complications in the pleura, chronic fistulas, rigidity, pleural adhesions, and fetid suppurations which make treatment long and difficult. In spite of this, patients with chest wounds do better than the abdominal cases. In several cases I have seen paraplegia from fracture of the spinal column associated with chest injuries.

*Casualties wounded in the head* by these big bombs rarely arrive alive in the hospital. But I have seen some who could be saved without major operation. The

only surgical treatment was excision of the edges of the wound, removal of damaged tissue and an aseptic dressing. Meningo-encephalitis is the most frequent cause of death.

Some patients recover without sequelæ but others develop complications due to meningeal adhesions and loss of cerebral substance.

The mortality in these cases is only less than that from abdominal injuries: that is, 66.8% of 112 cases treated in the Hospital General de Catalunya.

(2) The second type of bomb used in Barcelona was directed against the civilian population. These small light bombs explode horizontally with great force and velocity, and the appearance of the wounds differs completely from those produced by the heavy bombs. In some air raids in Barcelona, such as the one which took place on December 31, 1938, when many casualties occurred, this bomb was the only type employed.

Because of the great violence of the explosion (which may break windows more than one kilometre away), the contusion of all tissues, especially the nervous tissue, is very great, but without loss of consciousness. The patients feel no pain and one frequently sees great displacement in the fractures of the limbs, without any complaint on the patient's part. But it is not possible to take advantage of this insensibility, and to operate on the injured without anaesthesia, because the incisions are always painful. Before operating on such a patient it is necessary to improve his general condition, and what has given me best results is intense heating of the bed, especially by radiant heat. Blood transfusions, in small quantities (from 200 to 300 c.c.), and morphia are the best preparations before operation, combined with cardiac tonics.

The large number of little splinters into which the bomb was broken, produced small incisions in the skin which were always clean, painless and without hæmorrhage, on account of which, in many cases, they were missed if the patient was not examined completely undressed.

*Wounds in the legs*, of standing people, were the most frequent, because the little splinters have a horizontal projection. The lesions in the soft tissues especially in the arteries, nerves, and muscles, are very important and out of all proportion to the small entry wounds in the skin. The muscles are bruised and it is curious to observe the disproportion which exists between the size of the little splinters and the tunnel through the muscles. I think that this must be due to the great speed and spin which the splinter possesses.

One commonly sees severe compound and comminuted fractures. In the femur, I have seen several cases of fractures with great displacements and with lesions of the sciatic nerve produced by a splinter which was not greater than a finger nail in size.

It is necessary to operate on all the fractures caused by these small splinters although the minor importance of the lesion of the skin might induce one to treat these cases as if they were closed fractures. This mistake has produced several cases of death by septicæmia and especially by gas-gangrene. With an early and a rapid operation one can be conservative in many cases.

The experience of Catalonia in the Spanish War showed that treatment by closed plaster of Paris afforded a successful solution of all the problems which these patients present, especially problems of evacuation.

*Wounds of joints* are more grave, especially those which are not operated on in good time, but with correct technique it is possible to save many limbs, even though it is not uncommon to find articular stiffness or ankylosis as a sequel.

*Wounds of the abdomen* are usually penetrating, for which laparotomy is necessary. Frequently, with only one small wound in the abdominal wall one finds many intestinal perforations and even intestinal tears. As in all cases of perforation the prognosis depends on the time which has elapsed between the infliction of the wound and the operation, on the importance of the lesions, and on the level of the intestinal injuries. Wounds of the lower bowel are the more dangerous.

Very often the splinters produced wounds in the liver or spleen with grave hæmorrhage, demanding immediate operation. Rupture of the liver by a small splinter is very common. In the spleen total removal was sometimes necessary.

*In the chest* these splinters produce very small penetrating wounds which frequently cause severe hæmorrhage. All these wounds are "in the closed chest" and because of this it is better not to perform an operation. Immobilization of the chest, with rest and morphia for the patient, facilitates good results in many cases. Later major operations may have to be performed because of some complication such as deep-seated infection. In these patients the surgical technique is reduced to carrying out drainage. Only those cases in which there is grave hæmorrhage need immediate operation, and frequently lesions are found in the lungs which oblige one to perform partial pneumonectomies because of the existence of bruised tissues. These operations are badly tolerated by the injured, because they are in a state of profound shock before operation. I have not employed the high pressure room or similar apparatus, because in major wounds of the lungs, pulmonary insufflation produces more hæmorrhage, and it is best to allow a pneumothorax. In these cases blood transfusion is of value.

*In the skull*, when the splinters pass through it (and this they do unless they strike at a tangent) they produce a small perforation in the bone with a small irradiating fracture, never followed by depression. In the brain the splinter may lie deeply, sometimes being near the base of the cranium. This is because many civilians lie down on the floor and it is common to see splinters which have penetrated through the parietal bone localized in the base of the skull. Also in these cases wounds in the arms are frequent, since many people instinctively protect the head with the arms.

In these head wounds several expert examinations, neurological and radiological, are necessary, and as a general rule these patients should not be operated on in the casualty clearing hospital; their general condition permits an easy evacuation to a neurosurgical centre.

#### CASUALTIES PRODUCED BY FALLEN MASONRY

In this group injuries are of all kinds, and it is very difficult to classify them, but all these patients have some common characteristics which I shall describe. They are as follows:—

(1) The patients arrive at the hospital so completely covered by dust and earth as to be unrecognizable until they have been washed.

(2) All these injured are in a state of shock, but with special characteristics. I think it is a mixture of shock and concussion. They express their feelings with clearness, but with great indifference.

(3) They rarely present wounds, except a few in the head, but it is very common to see a skull depression without an external wound.

(4) Many of these patients have multiple injuries, with fractures, internal hæmorrhages, rupture of the liver, kidney or spleen, and collapse of the chest.

It is not often possible to operate on these casualties immediately because surgical intervention increases the primary shock, but fortunately in some cases as soon as shock diminishes with the appropriate treatment it is possible to perform the operations indicated.

## CLASSIFICATION OF THE CASUALTIES

After an air raid, all the people who have been injured arrive in the hospital in a very short time, in some cases together. For this reason their classification is the first and main point. If the work in the clearing room is appropriate and the selection of all cases is well carried out, it is possible to send many casualties to the base hospital or to their homes. I am able to give a classification but it is impossible to make a constant rule, since the patient is more important than the lesion, and similar injuries, in the same part of the body, may produce two quite different degrees of gravity.

*(a) Injuries Which Need Immediate Operation.*

- (1) Hæmorrhages.
- (2) Open chest wounds.
- (3) Great bruised destructions or avulsion of the limbs.
- (4) Small perforating abdominal wounds.

In this first group, only the patients with open chest wounds must remain in the hospital after operation. The others, in general, can be evacuated, especially after blood transfusion.

*(b) Injuries Which Need Immediate Operation as a General Rule.*

- (1) Closed depressions of skull.
  - (2) Major wounds in the head.
  - (3) Compound fractures produced by small splinters.
  - (4) Penetrating wounds in joints.
- (3) and (4) can be evacuated after operation. Evacuation of (1) and (2) depends on the patient's general condition.

*(c) Injuries Which Can be Sent to the Base Hospital Without Operation.*

- (1) Penetrating wounds in the skull, produced by small splinter-bombs.
- (2) Many cases of compound fracture, produced by small splinters, when hæmorrhage is unimportant, the patient is not shocked, and good immobilization is possible, provided the base hospital is not farther than two hours away. *It is important to compute the distance in time, not in miles.*
- (3) Many cases injured by fallen masonry, when they are not suffering from major shock.
- (4) Face wounds.

*(d) Injured Who Must Remain in the Clearing Station Without Operation.*

- (1) Those who present great shock.
- (2) Those with closed penetrating wounds in the chest.

*(e) Injured Who Can be Operated On if air raid causes few victims so that there are no other patients who need immediate surgical treatment.*

- (1) Those wounded in the abdomen by heavy bombs.

I have never seen cases of poison-gas, as this was never employed against our towns.

With these criteria it is possible to make a careful selection but a good classification must be done from the individual clinical data and especially according to the number of wounded and the facilities for work in a given clearing hospital.

## SUMMARY

I can draw the following conclusions :—

(1) Everything which facilitates the rapid transport of casualties to the hospital is of the utmost importance (brigades to extricate victims from the debris of fallen buildings and a well-organized and efficient ambulance service co-ordinated with the hospital service).

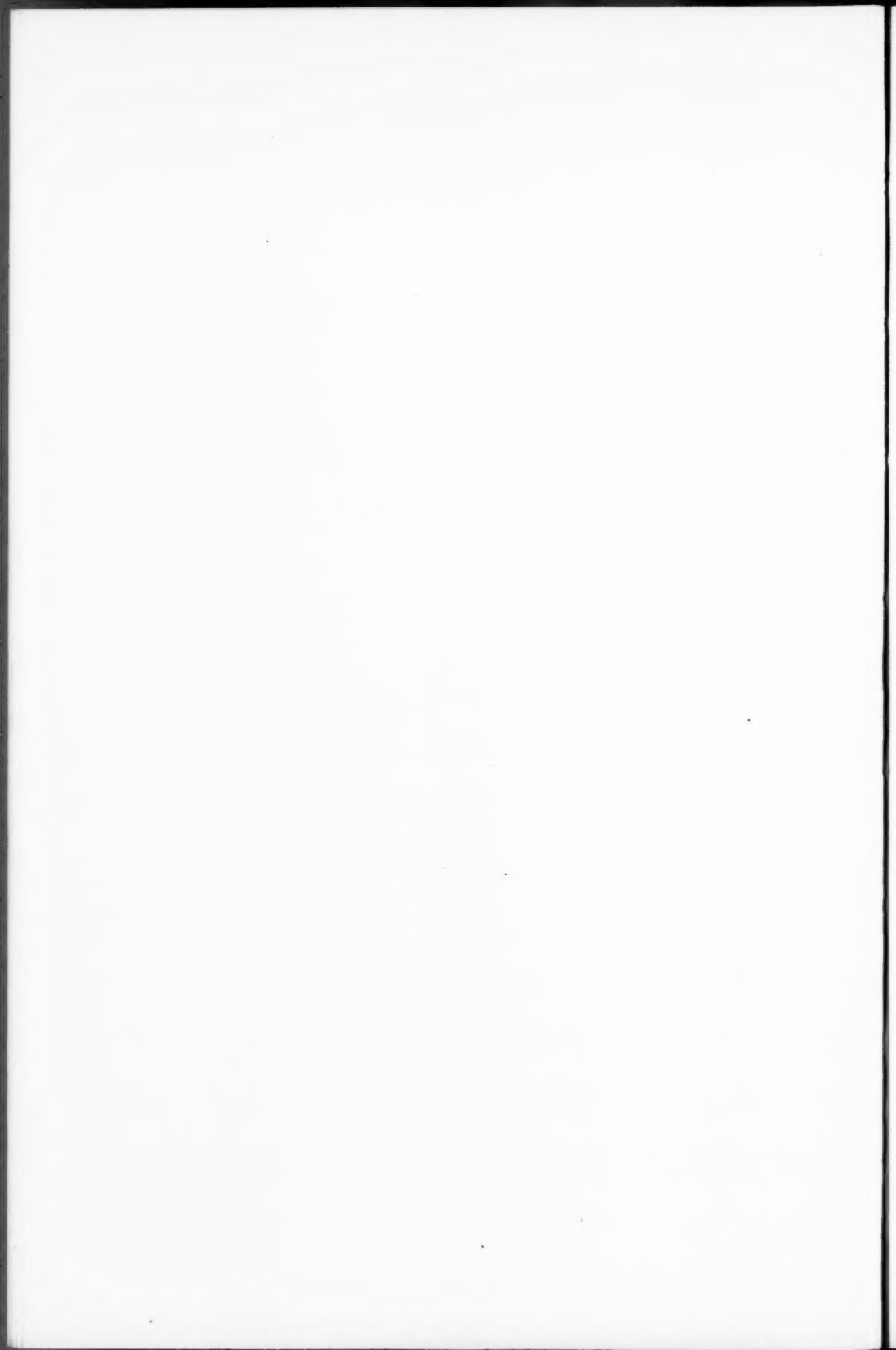
(2) The elimination of all unnecessary stages likely to delay the transport of wounded to the hospitals.

(3) Consequently my confirmed conclusion is insistence on the organization of so-called First Aid Posts only in connexion with the hospitals, and that such posts not forming part of a hospital should confine themselves to the treatment of minor superficial injuries and to the examination and reassurance of the nervous.

(4) All hospitals in the town should be organized, not merely for bandaging the wounded, but for the performance of such operations as are likely to be needed.

(5) Careful classification is a basis for a smoothly running organization.

(6) The operations should be carried out primarily on those whom it is possible to save. Instead of losing time on hopeless cases all one's energies should be concentrated on those who have a better chance of recovery.



## Section of Anæsthetics

President—HAROLD SINGTON, M.D., D.A.

[November 3, 1939]

### Yesterday and To-day in Children's Anæsthesia

#### PRESIDENT'S ADDRESS

By HAROLD SINGTON, M.D., D.A.

AFTER due deliberation, I have come to the conclusion that I could best serve the interests of the Section by a review of thirty years' experience in children's anæsthesia, with a description of how I found matters in 1907, the year I first went to the Hospital for Sick Children, Great Ormond Street; the general advances that were made, and the conditions that existed when my hospital work ceased. And I have secured the willing co-operation of my colleagues and successors, who will summarize the most recent methods practised in the beautiful modern operating theatres at Great Ormond Street, in the design of which for the anæsthetist's requirements I had considerable responsibility, and which have now been in use fifteen months. In 1907 it was considered an essential to the induction of anæsthesia in children that restraint should be forcibly exercised, so that before the anæsthetist approached the patient a nurse or nurses forcibly held the child, who was always in the supine position. Another feature was that post-anæsthetic vomiting was regarded as the invariable happening after an operation and was accepted as the natural sequel to an anæsthetic.

To-day the ease of induction and the almost total abolition of post-anæsthetic vomiting has made all the difference to the child, so that it is less often that a child objects to a second operation than an adult; and, in fact, it is rare for a child to mind in the least when, for instance, he or she visits the dental department for the second or third time for further dental extractions. To attain this end two elementary principles were observed:—

To be restrained physically and forcibly is objectionable to an adult and truly terrifying to a child. If you catch hold of a child, even when playing in the nursery, a struggle will result—it is a natural reflex. How much more terrifying forcible restraint must be when he is lying down, with a nurse or a student standing over him and using force. In such a case a struggle is *bound* to ensue. For this reason I established two axioms years ago—"Always have the child sitting up while anæsthesia is being induced" and—"Do not use restraint or have a nurse, or anyone else, within sight of the child". Of course in these pictures the children, being in the dental chair, will necessarily be sitting up, but it was the ease with which dental anæsthetics were induced, without the child showing any resentment, that made me realize the advantage to the child of the sitting posture, and originally influenced me to have all children sitting up in bed or on a couch, or

on the trolley, while I was inducing anaesthesia for other than dental operations—in fact for any operation. When sitting up there is no feeling of restraint and when no nurse is in sight there is no fear that the movements will be restrained by someone else. It is almost surprising at first how easy it all is. Sometimes the child may wave his hand, as if to get hold of the face-piece—his probable intention, but a word from the anaesthetist, if spoken in a persuasive manner, and not as a rough command, just—"Put your hand down"—and down will go the hand. You will see this in the film.

(Cinematograph film was then shown.)

Another point: I have found that induction by the open method is infinitely preferable with children, that is to say, with the anaesthetic on gauze in a Schimmelbusch or similar face-piece. The rubber-edged face-piece must never be pressed on to the face of the child while conscious, as it is a sensation that is resented and induces resistance, with inclination to struggle. Over thirty years ago in the dental department when we began to use ethyl chloride, it was given by the closed method, which necessitated a rubber face-piece, and also caused a feeling of suffocation; those were the days when anaesthesia was associated with a struggle and post-anaesthetic vomiting—two things which are now unknown.

I want to emphasize the great value of ethyl chloride and my conviction that it is particularly well suited for dental anaesthesia, in fact that it is more valuable than any other method when many teeth have to be extracted at one sitting, which is so frequently the case in hospital out-patients. It is completely safe—and it has been a puzzle to me why some schools have fought shy of its use; it is even taught in some institutions that ethyl chloride is a dangerous anaesthetic. In the dental department at Great Ormond Street Hospital it has been in use for thirty-five years, and from my own knowledge I can state that its administration has never given us the slightest anxiety. For thirty years of that period it was used exclusively; and only during the last five years have we used gas and oxygen occasionally for the older children; while for the last two years vinesthene has been given as a variant in some of the shorter administrations for the younger children.

The mistaken idea that ethyl chloride can be dangerous has arisen from ignorance of its action and failure to recognize the different stages of anaesthesia during administration. These stages are far more marked when it is given in a bag than when the open method is employed, and the second stage is characterized by tonic contraction of the masseters and pterygoids which clenches the jaw. This stage is synchronous with a short temporary period of cessation of respiration. If the golden rule of the anaesthetist has been followed—"to keep a patent airway"—the next breath of the patient further deepens anaesthesia and relaxation of the muscles of the jaw follows; but if the misguided anaesthetist tries to thrust forward the lower jaw during that period of fixation he must necessarily push forward the whole head, thereby narrowing the air-channel in the neck and consequently producing cyanosis. This cyanosis is not due to the ethyl chloride, but is caused by forcibly closing the airway. I believe that any anxiety that may have resulted from ethyl chloride anaesthesia is from this cause, and I feel compelled to blame the anaesthetist and not the ethyl chloride. When the ethyl chloride is given by the open method the rigidity I have described is not so pronounced, but that period of cessation of respiration occurs, and, of course, must be recognized as a natural phenomenon which should not surprise the administrator and cause him to panic.

In 1907 most major operations were performed under chloroform. It had actually been handed down from generation to generation, in textbook after textbook, that chloroform was particularly well tolerated by children, and that it was in fact the best anaesthetic for use in children's surgery, a theory which took a long time and considerable controversy to explode. Actually at Great Ormond Street in 1907 and 1908, 310 and 447 lb. respectively of chloroform were used and only 63 lb.

and 89 lb. of ether. This is in striking contrast to 1937 and 1938 when the amounts were 34 and 18 lb. chloroform, and 2,017 and 2,266 lb. ether; although there were some 2,000 fewer operations in the later years than thirty years previously.

AMOUNT OF ANÆSTHETIC USED.

	1907 lb.	1908 lb.	1937 lb.	1938 lb.
Chloroform .. .. .	310	447	34	18
Ether .. .. .	63	89	1,498	1,923
Ethyl chloride 60 c.c. tubes ..	218	180	2,017	2,266

OPERATIONS IN HOSPITAL.

	1907	1908	1937	1938
Out-patient and Casualty .. .. .	6,530	6,449	3,806	3,700
In-patients .. .. .	1,784	1,841	2,294	2,530
	8,314	8,290	6,100	6,230

That vomiting occurred after these chloroform administrations does not now cause us any surprise. But it took a lot of persuasion to convince others how much better it would be to use ether than chloroform. The change over came from the U.S.A. but it was a slow process, and, meanwhile, the anæsthetist who did a large amount of work in deep chloroform anæsthesia for the dissection of tonsils in those days had a life of anxiety, and I know of at least one who suffered from insomnia and an anxiety neurosis in consequence. Still, even when ether became generally accepted as the anæsthetic of choice for the majority of operations on children, and the necessary quantity of atropine had been gradually increased to that standard dosage which still prevails at Great Ormond Street Hospital, post-anæsthetic vomiting persisted in the majority of cases. At that time pre-anæsthetic starvation, or partial starvation, was the general practice, and the administration of aperients or purgatives was the accepted pre-operation procedure. It was the valuable work of Dr. R. S. Frew, who investigated the condition of acetonuria, which put us on the right lines. He found that 62% of all the children in the hospital (apart from diabetics) showed the presence of acetone in the urine during the three or four days following admission, and that the urine of the children between the ages of 2 and 6 years gave the strongest reaction. In all cases it cleared up within a week. Frew attributed the temporary acetonuria to the change in diet, as the hospital diet contained relatively less carbohydrates than the children had had at home, an opinion strengthened by the fact that those on an exclusive milk diet at home, which remained the same in hospital, were immune. In consequence Frew gave these children glucose on admission with the result that acetonuria did not occur. Realizing that the purgatives and starvation to which these children had been subjected prior to operation must cause carbohydrate starvation, these procedures were abolished, and every child before operation was given glucose in quantity, and the glucose was continued also after the operation, frequently by adding it to a rectal saline administration. This practice has been continued ever since. The difference which this made to the amount of vomiting after operation was quite dramatic.

From my remarks you will understand that my aim has been to make an operation for children an adventure associated with as little unpleasantness as possible, and to retain the child's trust and confidence, in order that the doctor should not be looked upon as the "bogey-man". Consequently, before the days of barbiturates, I tried further to ease the lot of the child by premedication with sedatives and narcotics. Some years ago I read a paper before this Section on

"Premedication by Paraldehyde in Children" (*Proceedings*, 22, 1197, Sect. Anæ., 29), in which I summarized those events which led up to its use and described the method used then, which has remained unaltered in technique to this day. I will only remark that I still hold the opinion that it is the most reliable form of premedication for children and is absolutely safe.

Premedication by paraldehyde is another factor which has influenced post-anæsthetic vomiting. Owing to the long sleep engendered by its use the child has eliminated the greater part of the absorbed ether before awakening; and to this I attribute the result that after premedication by paraldehyde in children, vomiting does not occur.

That intravenous anæsthesia and spinal analgesia have not yet been mentioned is because I am not at all keen on either procedure for children. In the days before the Great War I gave many children intravenous injections of alcohol, but these cases gave me considerable anxiety and I confess that I failed to find out the dosage which would at the same time ensure safety for the child and achieve a satisfactory anæsthesia from the surgeon's standpoint. The same opinion holds good for other intravenous injections in those days with children.

With regard to spinal analgesia, neither the method of approach nor the results proved satisfactory. In short, as far as my own experience goes and from the statistics of the results of these methods, it seems to me that inhalation anæsthesia is much better suited for children than an injection either into a vein or the spinal theca, an approach which the child invariably strongly resents.

Finally, I would not consider that I had performed my duty unless I made a final plea for simplicity. This is the Section before which innovations in our work should be described and encouraged, and even to-day we are very far off the last word in anæsthesia. No branch of medicine has made greater strides in recent years than ours, and it is to the younger and rising generation that we look to carry on the good work. My own experience covers the administration of over 100,000 anæsthetics, and, with very few exceptions, these have all been inhalation anæsthetics. That I have only had two deaths during this time—one was a pulmonary embolus, as the surgeon was sewing up a hernia in which the sac had contained the child's uterus and one tube and ovary, and the other was a sarcoma of both kidneys in a baby, with the whole of the undersurface of the diaphragm involved (it died before anæsthesia was established)—should be ample testimony that there is not a great deal wrong with the simple methods of the past.

## Section of Odontology

President—F. St. J. STEADMAN, L.R.C.P., M.R.C.S., D.P.H., L.D.S.

[October 23, 1939]

### The Teeth of the Australian Aborigines

#### PRESIDENT'S ADDRESS

By F. St. J. STEADMAN, L.R.C.P., M.R.C.S., D.P.H., L.D.S.

If we could segregate selected members of the community and keep them isolated for a long period we should see the effect of various diets and conditions of life upon their general well-being, especially on their teeth. But as man lives for a considerable time in comparison with some of the lesser animals, the life of one observer would not suffice to see the experiment progress very far: there would have to be a continuous series of investigators over several generations. But in Tasmania nature has made the experiment for us on a colossal scale, for she has isolated there a whole race of people for a vast period of time during which, so far as we know, there was no intercourse of any kind between them and the outside world.

In 1936 I examined in some detail the teeth of this now extinct race (Steadman, 1937). Tasmania was separated from the mainland by the foundering of Bass's Strait in the late Pliocene or early Pleistocene times, probably round about a million years ago, and during the whole of that vast period of time these Tasmanians have been living there entirely out of touch with human beings in any other part of the world, and, in fact, unconscious of their existence.

Briefly, I found that the Tasmanians had large well-developed arches almost entirely free from periodontal disease, and that caries was comparatively rare. It then occurred to me to compare these extinct Tasmanian Aborigines with the rapidly diminishing Central Australian Aborigines, who probably also had been isolated for many centuries, in order to ascertain whether there were marked differences between the teeth and jaws of the two peoples. If there were such differences, I wanted to see if their diet and mode of living could account for them.

For the purpose of my research I examined the 156 skulls which are at present in the Museum of the Royal College of Surgeons, and was at once struck by the large well-developed arches which were very similar to those of the Tasmanians. It has been said that the Tasmanian teeth are larger than those of any known race of men, but this statement seems to be incorrect, the jaws and teeth of the Australian Aborigines being equal in size to those of the Tasmanians. Indeed, in this regard there is a remarkable similarity between them, both having heavy, well-formed jaws, well-developed arches and large strong teeth, and both having facial bones, jaws and teeth considerably heavier and more massive than those to be found in the average modern European.



Among the Australian Aborigines I found the following :—

Total skulls examined	..	..	156	Total permanent teeth recorded	..	2,653
Total skulls with caries	..	..	18 (6)	Total permanent teeth with caries	..	62
Percentage	..	..	11.5	Percentage	..	2.3
Total teeth recorded	..	..	2,678	Total deciduous teeth recorded	..	25
Total teeth with caries	..	..	62	Total deciduous teeth with caries	..	0
Percentage	..	..	2.3	Percentage	..	0

1	2	2	1	3	4	1	2	0	0	2	5	2	3	3	1
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
0	1	3	2	2	2	0	1	1	1	1	2	0	6	2	6

The freedom from caries compared with civilized man is remarkable; and these figures are very similar to those found by Campbell in his examination of the aborigines of the Anmatjera and Ipirra tribes. His figures are as follows :—

Total individuals examined	..	73	Total permanent teeth recorded	..	1,844
Total individuals with caries	..	25	Total permanent teeth with caries	..	85
Percentage	..	34.2	Percentage	..	4.6
Total teeth recorded	..	2,098	Total deciduous teeth recorded	..	254
Total teeth with caries	..	89	Total deciduous teeth with caries	..	4
Percentage	..	4.2	Percentage	..	1.5

0	1	7	2	1	0	1	2	2	1	1	2	3	8	0	0
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
1	3	20	4	1	0	0	0	0	0	0	2	1	17	2	3

Campbell (1938) states that the dental caries among these tribes is in the main limited to aborigines in the later years of life. In all the cases of children that Campbell has examined, he only found three with caries. I found no caries among the children in either the Tasmanian or the Australian Aborigines. It is admitted, however, that the number examined is very small.

When caries is found, it often occurs, as one would expect, in several teeth in the same jaws. For example: Four teeth were carious in one skull (7), five in another (8), six in another and eleven in another (9); and, what is more remarkable, six of these last were among the lower front teeth.

There may have been multiple caries in other skulls, but one has to remember that in 57 of the 156 cases examined, the mandibles were missing; also a large number of the teeth were missing from the skulls, some before, but the majority after, death. Most of the molar teeth which were lost before death were probably lost by abscesses due to attrition.

Looking at Campbell's figures, the large number of first permanent molars which were carious is remarkable. In the figures quoted above, 52 out of 85 occurrences of caries affected the first molars, and of the 52, 37 affected the first lower molars.

In the Australian skulls examined by me, of the 62 instances of caries, only 14 affected the first permanent molars, nine of this 14 being in the lower: rather a striking difference for which it is difficult to account.

As with the Tasmanians, so with the Australians. The amount of attrition present in these skulls is almost incredible. Skull after skull showed marked attrition. Whole surfaces of the enamel on the molars had been removed, exposing the dentine, and even reaching the pulp cavities. Nearly all the adult bites were edge to edge,

because the mandible was able to slide forward, owing to the loss of the cusps of the teeth, and to take up an edge-to-edge position.

In the young adult of 20 years or so, where the attrition was not so marked owing to the short period of use, the edge-to-edge bite had not developed to the extent it had in older people. I should expect to find a normal bite in children, but unfortunately out of the seven children's skulls examined, six had no mandibles. In the skull with the mandible, both the upper and lower incisor teeth had been lost after death, and one had to work out the direction of these from the empty sockets (10). This marked attrition of the teeth, leading to irritation of the pulp, frequently caused local abscesses round the roots of the teeth.

As the mandible comes forward, there appears to be a flattening of the *eminentia articularis*, and it was noteworthy how flattened this was in some of the older people compared with the younger who had not yet developed the edge-to-edge bite.

In three skulls (11) the attrition was more marked on the left side in the canine region, leaving a gap between the occlusal surfaces of the affected teeth when the mouth is closed, similar to that occasionally found in the modern European, due to pipe smoking. Tobacco, as we know it, was of course unknown to these aborigines until the coming of the white man, and the attrition may have been due to some other deleterious habit.<sup>1</sup>

This marked attrition, as I have already pointed out in dealing with the Tasmanian skulls, is due to the nomadic habits of the tribes. The Tasmanian people lived under the wildest conditions, without proper shelter of any kind: they had not even learned to build houses, only to put up a few inadequate wind shelters. All their food was cooked in the open and was exposed to dust and sand storms. Those of us who had the pleasure of seeing Dr. Campbell's film (Cambridge, 1937), showing the life of the aborigines of Central Australia, will have observed how the dust frequently blew into their food. For some months during the Great War I was living on the Sinai Desert where one had the experience of sand blowing into the food, getting into one's mouth, and gritting on one's teeth.

Here again Campbell notes that attrition is less marked in those groups of natives who have lived more or less under civilized conditions, whereas the wear of the teeth is marked and commences at the very beginning of tooth functioning in those tribes which live in their natural environment.

While I was examining the Tasmanian skulls in 1936, the most surprising observation, in the 52 skulls which I examined, was the very high percentage of impacted third molars, in spite of the fact that the arches were large and well developed. I noticed no less than 14 impacted upper third molars in the 33 skulls in which the molar region was sufficiently perfect to enable me to make a diagnosis, and in the mandibles I found eight impacted third molars in 21 mandibles.

In view of this I turned eagerly to the Australian Aborigines to see whether they had a like number of impacted third molars. In the 149 skulls which had the upper molar region in good enough condition to make a diagnosis, I only found one right, and one doubtful left impaction (12); one right with the crown pointing outwards<sup>1</sup> (13); and in the mandibles, one vertical impaction of the right molar (14) and two horizontally impacted third molars (15).

That is to say, there were only three impacted third molars in 149 upper jaws and three impacted molars in 88 mandibles.

This difference in the number of impacted teeth in the two races is extraordinary. The jaws and the teeth are very similar, so that I could in no way account for this remarkable difference.

<sup>1</sup> There was a similar case, R.C.S. 1096, among the Tasmanians. A curious fact, as these cases are by no means common.

Apart from the impaction of the third molars, other familiar irregularities are noted, such as the apparent congenital absence of all four third molars in 20.3005 and again in 20.7193, and the absence of the upper third molars in 20.721 (the mandible of this skull being missing). I found no such case among the Tasmanians.

Missing left upper lateral incisors were noticed (16). This peculiarity was also noticed in one of the Tasmanian skulls.

Instanding lower premolars were also observed in one skull and, in the left only, in another (17).

In one specimen, a young female, the upper deciduous second molars were retained, and the first premolars were not fully erupted at the age of about 20 years (18).

In another specimen the outer alveolar plates on the right side had been removed after death, exposing the roots of the teeth. It was interesting to observe that the apex of the lower third molar was in the inferior dental canal (19).

One or two skulls showed the third molars smaller than the second (20). As a student, under the influence of Tomes, I was taught that this was due to modern civilization, the mastication of soft foods leading to the diminishing size of the jaws and, consequently, of tooth size.

Other peculiarities noticed were fan-shaped incisors (21), imbrication of lower incisors (22), also seen in the Tasmanian skulls, and two possible cysts.

Another interesting case was that of a fracture through the angle of the right side of the mandible which had no doubt healed under the expectant treatment without wiring or splinting (23).

Fifteen skulls showed one, generally the right, or both, upper central incisors removed for ceremonial purposes (24). In one case the missing incisor gap had closed (25). This is rather interesting because, in certain tribes, the reason given for the removal of these incisors is to prevent overcrowding and to make room for the remaining teeth. This ceremonial removal of the incisor teeth I found to be common in the Tasmanian Aborigines, occurring in nine out of the 52 skulls examined.

The interesting point is that it seems hardly likely that this curious custom should have sprung up concurrently in both races. It would seem to indicate that these races must have had a common origin or must have been in communication with people who practised this custom; but as we know that the Tasmanians had no communication with the outside world for over a million years, the origin of the custom would appear to be exceedingly ancient.

The life of a dental surgeon practising among these aborigines would be very different from anything within our experience. For one thing, he would have practically no periodontal disease to treat, or the cases he had would be very few and far between. How different from to-day when the majority of people are suffering from this disease. Again, caries would be infrequent, indeed would be so comparatively rare that he would be able to take his time over the treatment of the few carious teeth met with. Most astonishing of all, he would have no dentures to make. The number of teeth lost would be so few that he could forget all his knowledge of prosthetic dentistry without harming his patient.

He would, however, have a number of patients with swollen faces and acutely tender teeth due to abscesses caused by attrition; and he would find it advantageous to understand the art of practising local anaesthesia in removing the teeth, although most of them would be loose or lightly attached to the bone, owing to the acute periodontitis around them. He would also have to know how to remove impacted third molars, especially if he practised among the Tasmanians.

His chief difficulty would be to combat the vast amount of attrition to be met with among his patients, by instructing them to eat their meat under shelter. Indeed

we can imagine his experimenting with his patients by segregation of children carefully protected from dust, and he would note that little attrition would then occur; and he would discover that the edge-to-edge bite, with which he was so familiar, was not normal, and that the lower teeth should bite under the upper.

It would be difficult for an expert anthropologist to distinguish between the Tasmanian and Australian Aborigines if his examination were limited to jaws and teeth alone, the respective skull formations of the two races being dissimilar. The dental diseases also of the two races are very similar: there is a vast amount of attrition causing local abscesses, very little general periodontal disease, and much less caries than in civilized man of to-day.

When we recollect that these two distinct races were completely isolated from one another for a vast period of time, it is clearly well worth while to examine the conditions under which they lived in order to ascertain, if we can, why their respective dental diseases are so similar and yet so different from those of modern man.

Of the two races, the Tasmanians were the more backward. Their number in 1804 has been estimated at about seven or eight thousand. It is probable that this is about the maximum number that the Island, large though it is (about 24,331 square miles), could support, for nomad hunters require a large area on which to live.

These aborigines probably reached Tasmania by migration from New Caledonia and the neighbouring islands through a broken line or chain of islands now submerged, similar to the present chain between New Caledonia and New Guinea. They lived under the wildest conditions and must have been a hardy race. Exposed as they were to all weathers, they spent their lives wandering from place to place in search of food. The climate of Tasmania during part of the year would be harsh, and the aborigines, having no clothes and not having learned the art of the needle, and being without shelter, would lie naked, huddled together at night so close to their fires that severe burns would sometimes result.

At the time of the first settlement of white men in Australia in 1788, the Australians were probably more advanced than the Tasmanians ever became. It is estimated that their number at this date was about 300,000 divided into about 500 tribes, according to Chewings that number has now dwindled to 60,000. The number in each tribe varied from 100 to 1,500 and averaged five to six hundred.

As with the Tasmanians, the origin of the Australians is obscure. According to Elkin, "The available evidence appears to show that that they came from Southern India, moving thence to Ceylon, down to and around the Malay Peninsula, and, finally, to Australia" (Elkin, 1938). Chewings states that "Their distribution over the whole of the continent was probably an accomplished fact before the period of desiccation which now obtains over all the west-central portions; at any rate before the period had reached the stage when rivers like the Finke, the Hanson and the Lander had ceased to flow, or the freshwater lakes had become salt pans, or the sand dunes which extend over such huge areas were formed, or the giant marsupials had become extinct. . . . The present desiccation is thought to have followed on the last (Pleistocene) Ice Age" (Chewings, 1936).

The Australian Aborigines were in advance of the Tasmanian in that many, but by no means all, of their tribes knew the use of the boomerang. They were also capable of transporting water in skins or wooden troughs. The Tasmanians on the other hand, appear to have had no utensils by which they could store or carry water save in water bags of skin. Neither knew the art of boiling, though both had fires.

Both races were entirely improvident: they had no thought of the morrow, and never stored their food against scarcity. When certain foods and roots were in season, they ate them; when they were not, they went without. The chief subject

of conversation would naturally be food. When they could obtain enough they gorged themselves, but they could go for days without food or water.

They were complete "communists". They shared all food. Both sexes were taught from infancy the vital lesson that all food must be shared. Some hunters would be successful, others unsuccessful, and it was impossible for them to live in any other way than by sharing. Each had his or her right to a share: the man would take his portion of meat off the bone, then throw it over his head to his wife or wives, who would take their share before throwing it to the children. However hungry, each person took only his or her share and no more.

They had great powers of observation and knew the habits of every living thing around them, great and small. Their wonderful power of observation is illustrated by a story told by Dr. Chewings. On one occasion, when he was camping for the night, he spread his blanket near a hollow log. His boys (all native servants young or old are known as "boys") pointed out the track of a snake going towards the log, but no track going away from it. They insisted there was a snake in the log and set fire to it, when a large poisonous snake emerged.

On another occasion Chewings noticed his boys digging deep in the sand. They went down on their knees, dug a hole, arm deep, and brought up large inflated frogs. They would then squeeze the water from the frogs into their mouths. A small spot of sand which had been disturbed indicated where to dig.

The Australians, unlike the Tasmanians, had dogs, which arrived with them when they reached the country. These were useful in hunting, even if the game were only snakes, rats and lizards which were trailed by scent.

Both people ate anything which was edible. Campbell gives a brief list of foods eaten by the Australians as follows: Fluids, water, milk; plant foods, roots, tubers, bulbs, stems, leaves, fruits; cereals; aquatic and sea foods; flesh food; miscellaneous, such as honey, insects, infusions, eggs, &c. (Campbell, 1939).

Water was obtained not only from natural springs, the season's rainfall, &c., but from the roots of water-bearing plants and trees, as, for example, the water-bearing mallee (*Eucalyptus oleosa*). Since in such ways they could find a supply, they would frequently take long journeys without troubling to bring water with them, relying solely upon those sources.

The only milk they drank was from the human source. The women breast-fed their children up to the age of three years, or even longer. It is interesting to compare this consumption of milk during infancy only with the modern teaching. Milk is now given in many schools at the mid-morning break. Crawford and Broadley state "Abundant evidence is forthcoming from experiments conducted in schools, of the striking benefits conferred by the regular consumption of additional milk. Both height and weight have been increased beyond the average. The children taking milk at school have been shown to be less subject to illness, particularly nasopharyngeal catarrh. They suffer less from chilblains; their skins are in better condition; they are more alert and high spirited".

In the acquisition of food it was a recognized rule that the men hunted for game while the women gathered seeds, yams, fruits, and bulbs. Neither sex passed anything edible. Plant foods were numerous. They ate roots of the native carrot, young bean tree sapling, large yams, corms of the nut grass or yelka, which were dug up in season and consumed in large quantities. Fresh green leaves and stems for food were obtained from the munyeroo (*Portulaca oleracea*) and the well-known parakilja (*Calandrinia balonnensis*) and the leaves of the native lilac. Cereals such as seeds of grass and acacia and munyeroo were eaten when in season, raw or baked.

Campbell mentions the native fig (*Ficus platypoda*), the native peach (*Fusanus*

*acuminatus*), the native plum (*Santalum lanceolatum*). Fruits of the *Solanum*, which when ripe are like yellow gooseberries were much appreciated, also that of the pig-face (*Mesembryanthemum*) and the native cherry, the mistletoe (*Loranthus*) and other small berries were eaten when available.

Fish was speared and eaten in places where it could be obtained; when it was available the natives appeared to be fond of it. The Tasmanians also speared fish, but only for sport. Fishing with fish-hooks or nets was unknown. Molluscs, oysters, mussels, &c., were eaten when available. Campbell states that practically all available mammals were consumed as food. The larger ones, kangaroos and wallabies, were special favourites. They ate their dogs in times of stress. Rabbits and domestic cats have been added to the bill of fare in recent times since the coming of the white man. Birds such as the emu, wild turkey when available, crows and vultures, wild pigeons and similar birds were eaten. Also the larger non- or slightly poisonous reptiles, lizards, grubs and green caterpillars.

Campbell states that a certain amount of bone material was also eaten, the long bones being broken up with stones and the marrow then extracted. Fragments of the actual bone were also swallowed. On one occasion he observed a boy crunching up the bones of a cooked rabbit as if they were merely muscle. Eggs, when they could be obtained, of the emu, turkey, bustard, mallee fowl, were also eaten. Honey from the native bee was appreciated and hours would be spent chopping out the "sugar bag" from hollow trees, and deep holes were dug to get the honey ant. Honey-holding flowers were steeped in water and the juice expressed and drunk.

It is recorded that they also ate insects such as the bodies of moths filled with yellow oil, resembling in taste a sweet nut. Gum acacia and sugar tree manna were also eaten.

From this brief account of food it will be seen that their diet covered a wide range and was fairly well balanced.

In infancy they obtained the necessary protein (as well as fat, vitamins and salts) by the prolonged breast feeding which gave them such a good start in life. In later life protein was obtained from the flesh food. Fats were chiefly obtained from the same source. They consumed less carbohydrate food than civilized races. Sugar was scarce, the chief source being honey and manna from the sugar tree. A scanty supply of starch was obtained from cereals, tubers, and roots. Cleland and Johnston (quoted by Campbell) suggest that the large yams (*Ipomoea calobra*) "probably yield a considerable amount of starchy foods".

The most likely source of vitamin A, according to Campbell, was from the tissue fats and the livers of the various animals they consumed, also a certain amount from the occasional green foods and the few fruits and berries. Vitamin B, occurring in most food-stuffs in their natural state, was probably obtained from the cereals and greens and B<sub>2</sub> from meat, legumes, greens, and fruits. Vitamin C was probably obtained from the meats, fruits, and vegetable material. The source of vitamin D is doubtful as "it occurs more sparsely in natural food-stuffs than the other vitamins". It may have been obtained from the fat of milk, yolk of eggs or certain fish-liver oils, all items of food out of reach of many of the aborigines. Campbell suggests that the abundance of direct sunlight to which the aborigines were exposed may have supplied the necessary vitamin by generating it either in their skins or in the blood below the surface. As to the necessary salts, they were probably obtained as follows: calcium from the water, flesh food and vegetable matter, and particularly from the bone material as described by Campbell; phosphorus from flesh food, cereals and bone material and iron and iodine from the flesh food. As Campbell points out, the aboriginal's method of cooking his food with little loss of blood from the carcass ensured a rich supply of iron (Campbell, 1938).

Neither the Tasmanians nor the Australians boiled or stewed food, they had not the art nor any suitable utensils. A large part of their food was eaten raw or placed in an oven of hot sand and ashes. Corms, fruits, roots and stems were often cooked in this way. Seeds were ground between stones, mixed with water and eaten or baked in the form of a cake in the oven of sand and ashes. This method allowed a certain amount of sand to get into the food.

According to Campbell and Chewings the fur or hair of animals was first singed off in a blazing fire, then the charred hair was rubbed off by hand and the intestines removed.

Other rejected parts, e.g. fish scales, bird's feathers, &c., were removed and the carcase then placed in hot ashes covered with sand and so baked. The time of baking depended a good deal on the appetite of the cook and his friends. Frequently a very small amount of baking was done before the food was dragged out and eaten. This method of cooking, as Campbell points out, formed a hard crust on the surface of the carcase which preserved the fluids and salts. Indeed the method assures the greatest possible retention of the flesh constituents. Nothing was wasted: the entrails, after being emptied, were thrown into the hot embers and eaten with great relish. Chewings says: "They tear at their food like animals in a Zoo."

Chewings thus describes a meal which he witnessed: "A mob of natives was seen gathering, in great numbers, a green caterpillar, two or three inches long and thicker than a lead pencil in girth. They were in countless numbers and travelling on a broad front across a patch of green grass several acres in extent. Very voracious, they ate day and night, clearing the grass as they advanced. The native women, as they gathered them, pulled their heads off, held them by the tail and emptied them by drawing finger and thumb downwards. The remainder was then dried in hot ashes and eaten, or placed in a bag to dry. Later they were pounded lightly between stones, kneaded into a paste, and baked on hot stones. Whilst eating caterpillars they gave off an unpleasant odour from their bodies."

Food prepared in this primitive way, had on its surface a fair percentage of sand and grit, not only from the way it had been cooked, in sand ovens, but from the sand blown over it during the process. Chewings states that if a piece of meat happened to fall in the sand, they hit it against something to knock the sand off, or held it up between the finger and thumb of the left hand and gave it a flick with a finger of the right. When the husband had eaten what he wanted he threw it to his wife and children, who picked it up, knocked the sand off and ate.

This description, brief though it is, of the lives of these people (and it must be remembered that there is a considerable remnant of them living to-day and carrying on in precisely the same way) accounts for their comparative freedom from dental diseases. Indeed, those of the present-day tribes which have come in contact with civilization and have begun to eat some of the white man's food have been observed by Campbell to show a greater tendency to periodontal disease, and presumably to caries, than those who are still living in the natural state in the interior. Their food is never thoroughly cooked but eaten practically raw in circumstances which must allow a certain amount of sand to be taken into the mouth, thus accounting for the attrition.

We see that sugar is scarce, especially the easily fermentable variety, very much more scarce than is to be found in the diet of the modern white man. They have nothing comparable to the bread made from the fine white flour from which all the roughage has been carefully removed, cakes, pastries, jams, and stewed fruits to which sugar has been added, and other soft sticky carbohydrate food which clings to the teeth and gums causing so much havoc among the civilized white man of to-day.

It is hoped that this study will assist towards a better understanding of the

effects of diet on the dental conditions of civilized and uncivilized peoples respectively.

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- (3) R.C.S.M. No. 20.3003; 20.3551; 20.445; 20.5492; 20.6508; 20.6752; 20.7231; 20.7802; 20.82; 20.8555.
- (4) R.C.S.M. 20.2801; 20.3001; 20.3551; 20.4602; 20.61; 20.7345.
- (5) R.C.S.M. 20.3011.
- (6) R.C.S.M. 20.06; 20.0901; 20.1002; 20.1201; 20.2801; 20.2802; 20.29; 20.3004; 20.3951; 20.3954; 20.4602; 20.4603; 20.606; 20.6202; 20.6213; 20.8452; 20.8521; 20.8536.
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- (8) R.C.S.M. 20.06.
- (9) R.C.S.M. 20.2801.
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- (12) R.C.S.M. 20.3955; 20.6001.
- (13) R.C.S.M. 20.5455.
- (14) R.C.S.M. 20.1652.
- (15) R.C.S.M. 20.6765.
- (16) R.C.S.M. 20.4302.
- (17) R.C.S.M. 20.4202; 20.4401.
- (18) R.C.S.M. 20.5492.
- (19) R.C.S.M. 20.245.
- (20) R.C.S.M. 20.6211.
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- (25) R.C.S.M. 20.5493.

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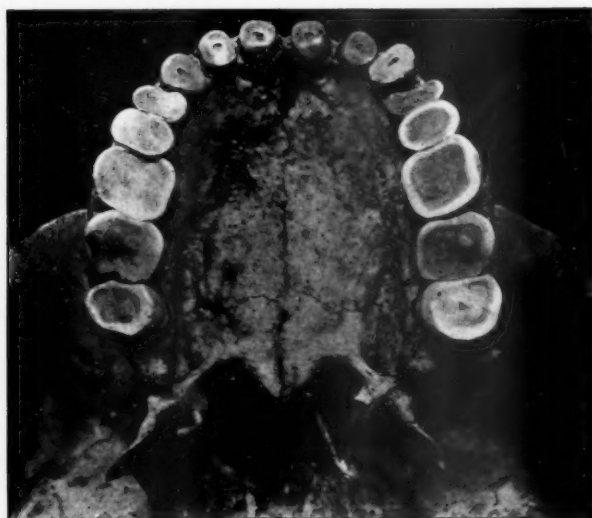
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R.C.S.M. No. 20.605 (actual size).



R.C.S.M. No. 20.605 (actual size).



R.C.S.M. No. 20.3007 (actual size). Showing advanced attrition.



R.C.S.M. No. 20.3007 (actual size). Showing advanced attrition.

## Section of Odontology

## AUSTRALIAN ABORIGINES

R.C.S.  
Museum

20.05



7	6	E	D	C	B	A	A	B	C	D	E	6	7
7	6	E	D	C	B	A	A	B	C	D	E	6	7

20.06



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

ci ic absc.

20.0801



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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No mandible

20.0802



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

x

20.0901



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

c absc.

most of the teeth appear to have been lost before death

20.0902



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

Marked attrition in 1 2 3 region Claypipe?

20.1001



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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No mandible

20.1002



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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absc. c x x x

No mandible

20.1201



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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absc. c c c

No mandible

20.123



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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in crypt in crypt

No mandible

20.1301



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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No mandible

20.1602



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

ci

20.1651



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
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No mandible

20.1652



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

23-25 Vimp.

20.1701



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

x

20.1702



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---

No mandible

20.1703



8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

in crypt c in crypt


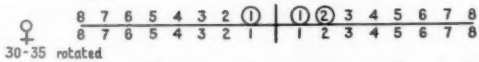
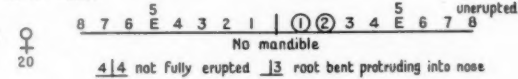
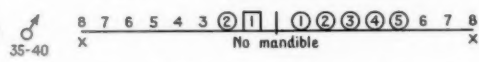
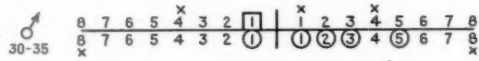
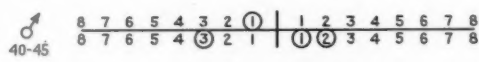
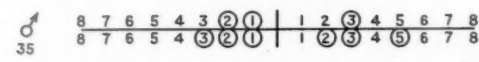
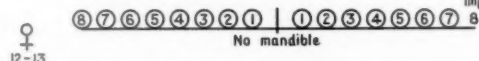
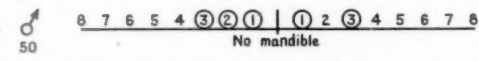
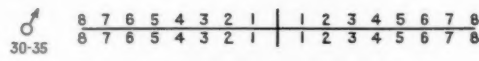
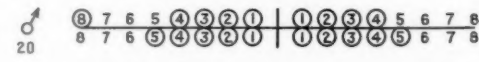
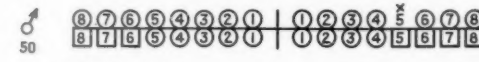
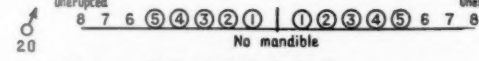
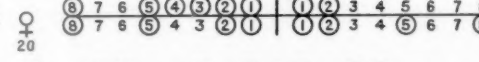
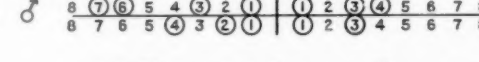
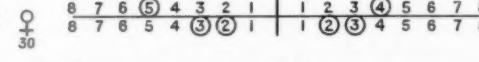
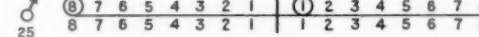
3-14 in crypt

crypt empty







- 20.5455   
 20.5491   
 20.5492   
 20.5493   
 20.5495   
 20.585   
 20.59   
 20.6001   
 20.6005   
 \* 20.605   
 20.606   
 20.61   
 20.6151   
 20.6152   
 20.6201   
 20.6202   
 20.6211 

\* Illustrated on p. 11

20.6212	♀ 20	8 7 6 (5) 4 3 (2) (1)   1 (2) 3 4 5 6 7 (8)	No mandible
20.6213	♂ 45	8 7 6 5 4 3 2 (1)   (1) 2 3 4 (5) 6 7 8 8 7 6 5 4 3 2 (1)   (1) 2 (3) (4) 5 6 7 8	3   pulp exposed (attrition) c <sub>a</sub>
20.625	♀ 40	8 7 6 (5) 4 (3) (2) (1)   (1) (2) (7) 4 5 6 7 (8) 8 7 6 5 (4) (3) 2 (1)   (1) 2 (7) 4 (5) 6 7 8	
20.6501	♀ 20	8 7 6 5 (4) 3 2 1   1 2 3 4 (5) 6 7 8 8 7 6 5 4 3 2 1   1 2 3 4 (5) 6 7 8	small small
20.6502	♀ 20 25	(8) (7) (6) (5) (4) (3) (2) (1)   (1) (2) (3) (4) (5) (6) 7 8	No mandible
20.6503	♂ 25	(8) 7 6 5 4 (3) (2) (1)   (1) (2) (3) (4) (5) 6 7 8	No mandible
20.6504	♀ 40	(8) 7 6 (5) (4) 3 (2) (1)   (1) (2) (3) (4) (5) 6 7 8	No mandible
20.6505	♂ 30	(8) 7 6 (5) (4) 3 (2) (1)   (1) (2) (3) 4 (5) 6 7 (8) 8 7 6 5 4 3 2 1   (1) (2) 3 4 5 6 7 8	absc. absc.
20.6506	♂ 30	unrupted, 8 7 6 5 4 3 (2) (1)   (1) 2 3 4 5 6 7 8 unrupted, ?	No mandible
20.6508	♂ 30	8 7 6 (5) 4 3 2 (1)   (1) 2 3 4 5 6 7 8 8 7 6 5 4 3 2   1 2 3 4 (5) (6) (7) 8	
20.6510	♂ 50-55	8 7 6 5 4 3 2 (1)   (1) 2 3 4 5 6 7 8? 8 (7) 6 (5) (4) 3 2 (1)   (1) 2 (3) 4 (5) 6 7 8?	roots curved back absc.
20.654	♂ 35	(8) (7) 6 (5) (4) (3) (2) (1)   (1) (2) (3) (4) (5) 6 (7) (8) (8) 7 6 (5) (4) (3) (2) (1)   (1) (2) (3) (4) (5) 6 7 8	
20.656	♂ 30	(8) 7 6 5 4 3 (2) (1)   (1) (2) (3) 4 5 6 7 8	No mandible
20.66	♂ 35	Periodontal disease (8) (7) 6 (5) (4) 3 (2) (1)   (1) (2) (3) 4 (5) (6) (7) (8) 8 7 6 (5) (4) (3) (2) (1)   (1) (2) (3) (4) (5) (6) 7 8	Periodontal disease
20.67	♀ 7	(7) 6 E D C (B) (A)   (A) (B) (C) D E 6 (7)	No mandible
20.6751	♂ 55	(8) 7 6 (5) (4) (3) (2) (1)   (1) 2 (3) 4 5 6 7 8	No mandible
20.6752	♂ 25	(8) 7 6 5 4 3 (2) (1)   (1) 2 3 4 5 6 7 (8)	No mandible
20.6754	♂ 30-40	(8) (7) 6 (5) (4) (3) (2) (1)   (1) (2) (3) (4) (5) 6 (7) (8) 8 7 6 (5) (4) (3) (2) (1)   (1) (2) (3) (4) (5) 6 7 8	

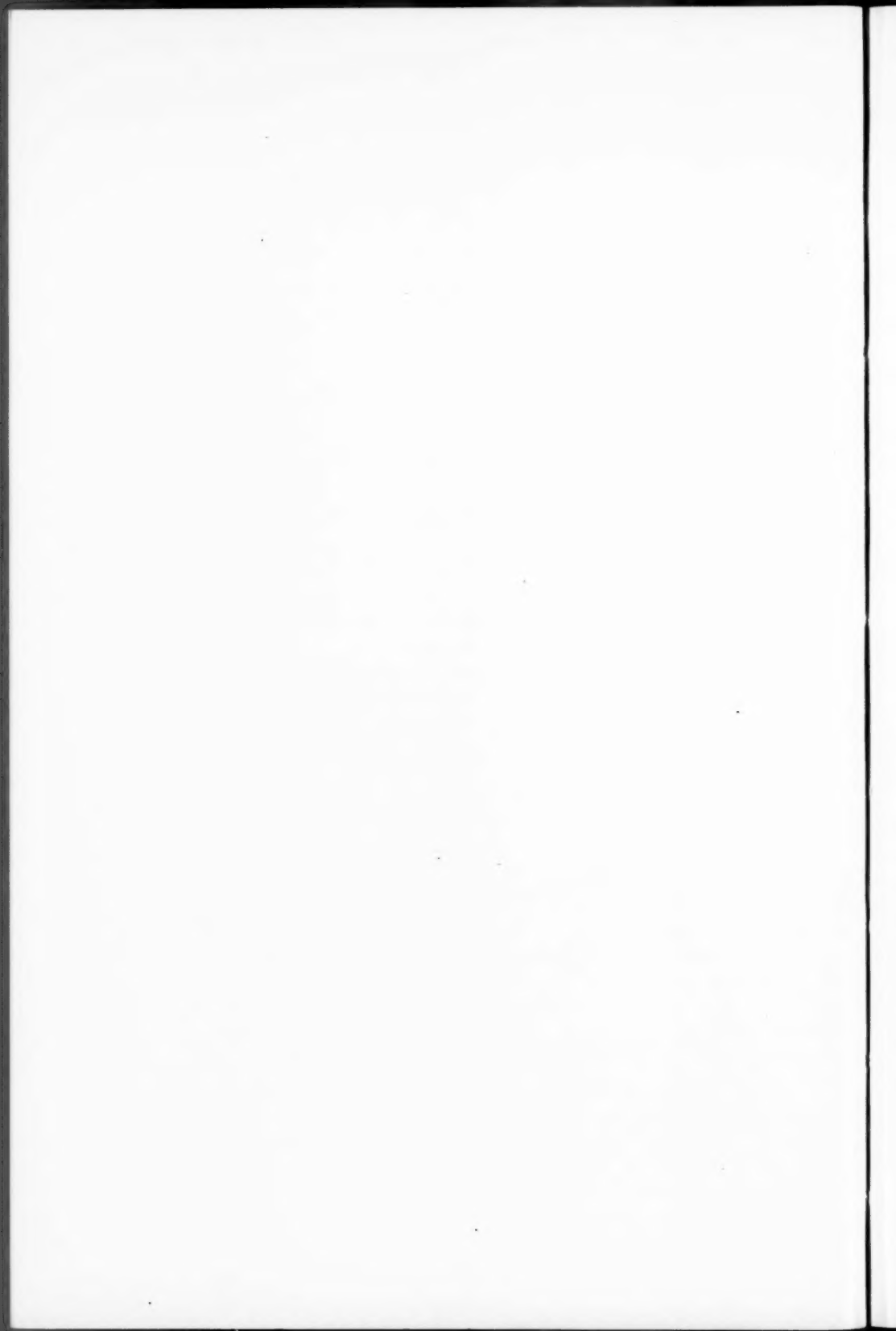
- 20.676 ♂  
30-35
- |   |   |   |     |     |     |     |     |  |     |     |     |     |     |   |   |   |
|---|---|---|-----|-----|-----|-----|-----|--|-----|-----|-----|-----|-----|---|---|---|
| 8 | 7 | 6 | (5) | (4) | (5) | (2) | (1) |  | (1) | (2) | (3) | (4) | (5) | 6 | 7 | 8 |
| 8 | 7 | 6 | (5) | (4) | (3) | (2) | (1) |  | (1) | (2) | (3) | (4) | (5) | 6 | 7 | 8 |
- 20.6765 ♂  
30-35  
imp.
- |   |   |   |     |     |                |                |     |  |     |     |     |     |     |   |   |   |
|---|---|---|-----|-----|----------------|----------------|-----|--|-----|-----|-----|-----|-----|---|---|---|
| 8 | 7 | 6 | (5) | (4) | <sup>x</sup> 3 | <sup>x</sup> 2 | (1) |  | (1) | (2) | 3   | (4) | (5) | 6 | 7 | 8 |
| 8 | 7 | 6 | (5) | (4) | (3)            | (2)            | (1) |  | (1) | (2) | (3) | (4) | (5) | 6 | 7 | 8 |
- 20.7150 ♂  
25
- |     |     |     |     |   |   |     |     |  |     |     |   |   |     |   |   |     |
|-----|-----|-----|-----|---|---|-----|-----|--|-----|-----|---|---|-----|---|---|-----|
| (8) | (7) | (6) | (5) | 4 | 3 | (2) | (1) |  | (1) | (2) | 3 | 4 | (5) | 6 | 7 | (8) |
|-----|-----|-----|-----|---|---|-----|-----|--|-----|-----|---|---|-----|---|---|-----|
- No mandible
- 20.7172 ♀  
45-50
- |   |   |     |     |     |     |     |     |  |     |     |   |     |   |     |   |   |
|---|---|-----|-----|-----|-----|-----|-----|--|-----|-----|---|-----|---|-----|---|---|
| 8 | 7 | 6   | 5   | 4   | 3   | (2) | (1) |  | (1) | (2) | 3 | (4) | 5 | 6   | 7 | 8 |
| 8 | 7 | (6) | (5) | (4) | (3) | (2) | (1) |  | (1) | (2) | 3 | 4   | 5 | (6) | 7 | 8 |
- 20.7191 ♂  
45
- |   |   |   |   |   |   |   |   |  |   |   |   |     |   |   |   |   |
|---|---|---|---|---|---|---|---|--|---|---|---|-----|---|---|---|---|
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |  | 1 | 2 | 3 | (4) | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|--|---|---|---|-----|---|---|---|---|
- No mandible
- 20.7192 ♀  
35-40
- |     |     |   |   |   |   |   |     |  |     |     |     |   |   |   |   |   |
|-----|-----|---|---|---|---|---|-----|--|-----|-----|-----|---|---|---|---|---|
| (8) | (7) | 6 | 5 | 4 | 3 | 2 | 1   |  | (1) | (2) | (3) | 4 | 5 | 6 | 7 | 8 |
| 8   | 7   | 6 | 5 | 4 | 3 | 2 | (1) |  | 1   | 2   | 3   | 4 | 5 | 6 | 7 | 8 |
- 20.7193 ♀  
35
- |   |   |   |   |     |     |     |     |  |     |     |                |     |     |   |   |   |
|---|---|---|---|-----|-----|-----|-----|--|-----|-----|----------------|-----|-----|---|---|---|
| ? | 7 | 6 | 5 | 4   | 3   | (2) | (1) |  | (1) | (2) | <sup>x</sup> 3 | (4) | 5   | 6 | 7 | ? |
| ? | 7 | 6 | 5 | (4) | (3) | 2   | (1) |  | (1) | 2   | (3)            | (4) | (5) | 6 | 7 | ? |
- Congenital absence of third molars
- 20.721 ♂  
50
- |   |   |   |   |   |   |   |   |  |   |   |   |   |   |   |   |   |
|---|---|---|---|---|---|---|---|--|---|---|---|---|---|---|---|---|
| ? | 7 | 6 | 5 | 4 | 3 | 2 | 1 |  | 1 | 2 | 3 | 4 | 5 | 6 | 7 | ? |
|---|---|---|---|---|---|---|---|--|---|---|---|---|---|---|---|---|
- No mandible  
Congenital absence of 8 | 8 ?
- 20.7231 ♂  
40-45
- |   |   |   |   |   |   |   |     |  |     |     |   |     |   |   |   |   |
|---|---|---|---|---|---|---|-----|--|-----|-----|---|-----|---|---|---|---|
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1   |  | (1) | (2) | 3 | (4) | 5 | 6 | 7 | 8 |
| 8 | 7 | 1 | 5 | 4 | 3 | 2 | (1) |  | 1   | 2   | 3 | 4   | 5 | 6 | 7 | 8 |
- 20.7232 ♀  
40
- |     |     |     |     |     |     |     |     |  |     |     |     |     |   |   |   |     |
|-----|-----|-----|-----|-----|-----|-----|-----|--|-----|-----|-----|-----|---|---|---|-----|
| (8) | (7) | (6) | (5) | (4) | (3) | (2) | (1) |  | (1) | (2) | (3) | 4   | 5 | 6 | 7 | (8) |
| 8   | 7   | 6   | 5   | 4   | (3) | 2   | (1) |  | (1) | (2) | (3) | (4) | 5 | 6 | 7 | 8   |
- 20.7251 ♂  
50-55
- |   |   |   |   |   |   |   |   |  |   |   |   |   |   |   |   |   |
|---|---|---|---|---|---|---|---|--|---|---|---|---|---|---|---|---|
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |  | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |  | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
- Edge to edge bite
- 20.7252 ♂  
45
- |     |   |   |   |   |   |   |     |  |     |     |     |     |     |     |     |     |
|-----|---|---|---|---|---|---|-----|--|-----|-----|-----|-----|-----|-----|-----|-----|
| (8) | 7 | 6 | 5 | 4 | 3 | 2 | (1) |  | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|-----|---|---|---|---|---|---|-----|--|-----|-----|-----|-----|-----|-----|-----|-----|
- No mandible
- 20.728 ♂  
25-30
- |   |   |   |   |     |     |     |     |  |     |     |     |     |     |   |   |   |
|---|---|---|---|-----|-----|-----|-----|--|-----|-----|-----|-----|-----|---|---|---|
| 8 | 7 | 6 | 5 | (4) | (3) | 2   | (1) |  | (1) | (2) | (3) | (4) | (5) | 6 | 7 | 8 |
| 8 | 7 | 6 | 5 | (4) | (3) | (2) | (1) |  | (1) | (2) | (3) | (4) | (5) | 6 | 7 | 8 |
- 20.7301 ♀  
40-45
- |   |   |   |     |     |     |     |     |  |     |     |     |     |     |   |   |   |
|---|---|---|-----|-----|-----|-----|-----|--|-----|-----|-----|-----|-----|---|---|---|
| 8 | 7 | 6 | (5) | (4) | (3) | (2) | (1) |  | (1) | (2) | (3) | (4) | (5) | 6 | 7 | 8 |
|---|---|---|-----|-----|-----|-----|-----|--|-----|-----|-----|-----|-----|---|---|---|
- No mandible
- 20.7306 ♂  
45
- |   |   |   |   |   |   |     |     |  |     |     |     |     |   |   |   |     |
|---|---|---|---|---|---|-----|-----|--|-----|-----|-----|-----|---|---|---|-----|
| 8 | 7 | 6 | 5 | 4 | 3 | (2) | (1) |  | (1) | (2) | 3   | 4   | 5 | 6 | 7 | (8) |
| 8 | 7 | 6 | 5 | 4 | 3 | 2   | 1   |  | (1) | (2) | (3) | (4) | 5 | 6 | 7 | 8   |
- absc due to attrition      absc (same)
- 20.7341 ♂  
35-40  
cyst.
- |   |   |   |     |   |   |     |     |  |     |   |   |   |     |     |     |     |
|---|---|---|-----|---|---|-----|-----|--|-----|---|---|---|-----|-----|-----|-----|
| 8 | 7 | 6 | (5) | 4 | 3 | 2   | 1   |  | 1   | 2 | 3 | 4 | (5) | (6) | 7   | (8) |
| 8 | 7 | 6 | 5   | 4 | 3 | (2) | (1) |  | (1) | 2 | 3 | 4 | (5) | (6) | (7) | 8   |
- absc      absc (a)      absc
- 20.7342 ♂  
20-22  
(inst.)
- |   |   |   |   |   |   |   |   |  |   |   |   |   |   |   |   |   |
|---|---|---|---|---|---|---|---|--|---|---|---|---|---|---|---|---|
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |  | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 8 | 7 | 6 | 5 | 4 | 3 | 2 | 1 |  | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
- damaged (inst.)      (inst.)      damaged
- 20.7345 ♀  
25
- |   |   |   |   |   |   |     |     |  |     |     |   |   |   |   |   |   |
|---|---|---|---|---|---|-----|-----|--|-----|-----|---|---|---|---|---|---|
| 8 | 7 | 6 | 5 | 4 | 3 | (2) | (1) |  | (1) | (2) | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|-----|-----|--|-----|-----|---|---|---|---|---|---|
- No mandible

- 20.7348 ♂  
35  
8 7 6 5 4 3 2 ① | ① 2 3 4 5 6 7 ⑧  
No mandible
- 20.7349 ♀  
20-25  
8 7 6 5 4 ③ ② ① | ① ② 3 4 5 6 7 8  
No mandible
- 20.7350 ♂  
45-50  
8 7 6 5 4 3 2 1 | 1 2 3 4 5 6 7 8  
8 7 6 5 4 3 2 1 | 1 2 3 4 5 6 7 8
- 20.7355 ♀  
30-35  
8 7 6 5 4 ③ ② 1 | ① 2 3 4 5 6 7 8  
8 7 6 5 4 3 ② ① | ① ② ③ 4 ⑤ 6 7 8
- 20.761 ♂  
35  
8 7 6 5 4 3 ② ① | ① 2 3 4 5 6 7 8  
8 7 6 5 4 3 ② ① | ① ② ③ ④ 5 6 7 8
- 20.7701 ♀  
35  
8 7 6 5 ④ 3 2 ① | ① 2 ③ 4 ⑤ 6 7 8  
No mandible
- 20.7702 ♂  
35  
8 7 6 5 4 3 2 ① | ① ② 3 4 5 6 7 8  
8 7 6 5 4 3 2 1 | 1 2 3 4 5 6 7 8  
Overcrowded mandible 2/2 behind 1/1
- 20.7751 ♂  
35-40  
8 7 6 5 ④ 3 2 1 | 1 2 3 4 ⑤ 6 7 8  
8 ⑦ 6 ⑤ 4 3 ② ① | 1 2 ③ ④ ⑤ 6 7 8
- 20.7752 ♀  
45  
8 7 6 5 4 3 ② ① | ① <sup>x</sup> 2 ③ 4 ⑤ 6 7 8  
No mandible
- 20.7801 ♂  
40-45  
8 7 ⑥ 5 <sup>x</sup> ④ 3 2 1 | ① 2 3 4 5 6 ⑦ ⑧  
No mandible
- 20.7802 ♀  
45  
⑧ ⑦ 6 5 4 3 2 1 | ① ② 3 4 5 6 7 8  
8 ⑦ ⑥ 5 4 3 2 1 | 1 2 3 4 5 ⑥ ⑦ ⑧  
absc.
- 20.79 ♂  
25-30  
8 7 6 5 4 3 2 1 | ① 2 3 4 5 ⑥ ⑦ 8  
8 7 6 5 4 3 2 1 | 1 2 3 4 5 6 7 8  
C1
- 20.811 ♀  
40-45  
8 7 6 5 4 ③ ② ① | ① <sup>x</sup> 2 <sup>x</sup> 3 4 5 6 7 8  
8 7 6 5 4 3 ② ① | ① ② ③ ④ 5 6 7 8
- 20.82 ♀  
20-25  
8 7 6 5 4 3 2 1 | 1 2 3 4 5 6 7 ⑧  
8 7 6 5 ④ 3 2 1 | 1 2 3 4 5 6 ⑦ ⑧  
Tumour?
- 20.8451 ♂  
40  
8 7 ⑥ 5 4 3 2 1 | 1 2 7 ④ 5 6 7 8  
⑧ 7 6 5 4 3 2 1 | 1 2 7 4 5 6 7 8  
57 | 67 Loss of bone cause
- 20.8452 ♀  
25  
8 7 6 ⑤ ④ 3 2 1 | 1 2 <sup>x</sup> ③ 4 5 6 7 8  
8 7 6 5 4 3 ② ① | 1 2 ③ ④ 5 6 7 8  
C1
- 20.8453 ♂  
35  
8 7 6 5 4 3 2 ① | ① ② ③ ④ <sup>x</sup> 5 6 7 ⑧  
No mandible
- 20.8454 ♀  
30  
⑧ ⑦ 6 5 4 3 2 1 | ① 2 3 4 5 6 ⑦ ⑧  
No mandible

20.8455	♀	in crypt	8 7 6 5 4 3 ② ①		① ② ③ 4 5 6 7 8	in crypt
		20 in crypt	8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	in crypt
			Edge to edge bite			
20.8456	♂		8 7 6 ⑤ ④ ③ ② ①		① ② ③ ④ 5 6 7 8	
		50-55	No mandible			
20.8461	♀		8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	
		40-45				ci
20.8462	♂		x 8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	
		20-25	8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	
			Developing edge to edge bite			
20.8521	♂		8 7 6 5 4 3 2 ①		1 2 3 4 5 6 7 8	
		45	8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	ci absc.
20.8522	♀		8 7 6 ⑤ ④ ③ ② ①		① ② 3 4 5 6 7 8	
		35	No mandible			
20.8536	♂		8 7 6 5 4 3 ② 1		1 2 3 4 5 6 7 8	absc. c
		30	8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	
20.8538	♀		8 7 6 ⑤ 4 3 ② 1		1 2 3 4 5 6 7 8	
		40	8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	
			Edge to edge bite			
20.8539	♂		8 7 6 5 ④ ③ ② ①		① ② ③ ④ ⑤ 6 7 8	
		45	8 7 6 5 4 ③ ② ①		① ② ③ ④ ⑤ 6 7 8	
20.8550	♂		⑧ 7 6 5 4 3 2 ①		① ② 3 4 5 6 7 8	
		45	8 7 6 5 4 3 2 ①		1 ② 3 4 5 6 7 8	Periodontal disease
20.8552	♂		8 7 6 ⑤ 4 3 2 1		1 ② 3 4 5 6 7 8	
		50	8 7 6 5 ④ 3 2 1		1 2 3 4 ⑤ 6 7 8	
			Crowded arches			
20.8554	♀?		in crypt 8 7 6 5 4 3 2 1		1 2 3 4 5 6 7 8	ci in crypt
		18	No mandible			
20.8555	♀		in crypt 8 7 6 ⑤ 4 ③ ② ①		① ② 3 ④ ⑤ 6 7 8	in crypt
		22 in crypt	8 7 6 ⑤ 4 ③ ② ①		1 ② 3 ④ ⑤ 6 7 8	in crypt
20.8561			⑧ ⑦ 6 5 4 3 2 1		① 2 3 4 5 6 ⑦ ⑧	
			8 ⑦ ⑥ 5 4 3 2 1		1 2 3 4 5 ⑥ 7 8	
			Narrow			

## EXPLANATION OF SYMBOLS

□ = tooth lost before death	C = caries
① = tooth lost after death	c̄ = occlusal caries
x = root	ci = distal
absc. = abscess	iC = mesial caries
inst. = instanding	c <sub>a</sub> = buccal caries
imp. = impaction	V. imp. = vertical impaction



## United Services Section

President—Surgeon Captain J. G. DANSON, Royal Navy

### On Injuries to the Brain and their Sequelæ

#### PRESIDENT'S ADDRESS

By Surgeon Captain J. G. DANSON, M.D., F.R.C.P. (Lond.),  
Royal Navy

[Owing to war conditions this address was not read before the Section at date of going to press]

THE Great War and the ubiquitous motor car have revolutionized our outlook on head injuries, so that for the past two decades less attention has been paid to fractures of the skull, and more to damage of the intracranial contents. Indeed in a series of 441 cases of head injury Maclure and Crawford found 39% of them to be free from fracture of the skull [1]. Except in cases of compound fractures, penetrating gunshot wounds, that rare form of extradural hæmorrhage due to injury of the middle meningeal vessels, and, less urgently, in cases of depressed fracture causing irritation, the modern trend is towards non-interference; and, whilst combating the risks of sepsis, the surgeon tries to assess the cerebral damage.

Trotter [2] has observed that "kinetic energy (on which the seriousness of accidents obviously depends) varies with the mass and the *square* of the velocity of the moving bodies concerned". Thus if an accident occurs in horse traffic moving at 10 miles per hour, and a motor accident occurs when moving at 30 miles per hour, the kinetic energy involved in the latter case is not three, but nine times that of the former. The same law applies to the bullets of war-time and the blasts of mines and high explosive missiles.

*Concussion.*—In all head injuries of any severity there is an initial stage, known clinically as "concussion". This may be a momentary stunning of the patient, or something longer. Trotter [3] defines it as "an essentially transient state due to head injury, which is of instantaneous onset, manifests widespread symptoms of a paralytic kind, does not, as such, comprise any evidence of structural injury, and is always followed by amnesia for the actual moment of accident".

No one has challenged Trotter's postulates for the state of concussion, but several have dissented from his view as to its causation. Following Kocher, he thought it was due to cerebral anæmia, the result of sudden deformation of the skull at the moment of accident. This view has been endorsed by several authorities, but

Russell [4] believes that there must be some molecular disturbance of the brain tissues themselves which inhibits the flow of impulses, the "commotio" in fact of the older writers. Jefferson [5] hints at an interruption of the higher neuronal mechanisms, the possibility of electrolytic disturbances, and of variations in the hydrogen-ion content of the brain tissues. Greenfield [6] rejects the vascular view as expounded by Trotter on several grounds; among others, that there is no evidence that this emptying of capillaries, if it does in fact occur, would cause a sudden cessation of the functioning of nerve cells. Anoxæmia would have to be continuous for some seconds to cause a general loss of nerve function. Incidentally, he also rejects the so-called "humoral" theory put forward by Duret [7]; that is to say, that at the moment of impact a wave of cerebrospinal fluid passes suddenly throughout the ventricular system, bruising and tearing as it goes, insulting all the structures within its reach, for instance the choroid plexuses, the thalamus, the hypothalamus, and the brain-stem. Attractive as he admits this theory to be, there is no evidence that this wave actually occurs; and even if it did, it would not explain hæmorrhages remote from the immediate course of the wave, such as those into the ventral portion of the pons. Greenfield, basing himself on histological research, inclines to the theory of the deformation and stretching of nerve elements and vascular tissues, due to sudden alterations in the shape of the brain, not only at the point of impact, but at any point where the movement of the organ is resisted, for example near the dural septa and the bony ridges on the floor of the skull. Although the lesions are widely spread they are definitely focal. Consciousness appears to be centred in the thalamus; and the sudden deformation or stretching of fibres leading to and from this region, would explain the loss of consciousness. It seems probable, he tells us, that this may be due to a sudden displacement of the cerebral hemispheres in relation to the incisura tentorii, distorting the connexions of the hemispheres with the brain-stem.

A proportion of fatal cases of concussion show few, if any, local lesions at autopsy; whilst others with gross and diffuse cerebral lesions had experienced only mild concussion in life. It is important to remember that in all but the mildest cases, concussion is accompanied by contusion, and possibly by other serious conditions as well. There has been a tendency accordingly to discard the word "concussion" altogether; but, since it is the symbol of a state which we can recognize clinically, it is useful to that extent, and should be retained accordingly.

In concussion the disturbance of function varies in severity and duration. The patient may be momentarily stunned, a degree of mental confusion and headache following, and the pulse and respiration remaining normal. The symptoms clear up within twenty-four hours. We see this sort of things in boxers; and the footballer may continue his game after an accident in an automatic fashion, "poorly adapted to its changing circumstances. Behaviour of this sort has always the stamp of imperfect conscious guidance" [8]. Such a state of affairs may follow a glancing blow, the cerebral hemispheres alone being affected; but concussion may be a much deeper and more serious matter than this, if the stem of the brain and the medullary centres are involved. There may be such severe impairment of bulbar function as to arrest respiration for the nonce. In fatal cases of this sort bulbar anæmia plays its part.

When a patient is deeply concussed, he lies limp and pale, his pupils are dilated and may be fixed. Except in speedily fatal cases where the pulse is rapid and feeble, its rate is usually slow, and respiration is shallow with periodic sighing. The reflexes are almost or completely gone. The respiratory centre fails first. As a rule, however, the pulse continues to beat; and the respirations, irregular at first, soon become deeper and better sustained. The reflexes return, the reaction to light being the first to appear. The patient is restless and irritable, and vomits

frequently. The blood-pressure improves, and the temperature may be slightly raised, the patient complaining of headache. Up to this point the lower and vital mechanisms are recovering their functions. The restoration of the higher functions, as perceived clinically, will depend to a considerable extent on the patient's personality. He may be irritable or emotional, talkative or drowsy, but he will not be reserved. Later, the normal inhibitions and insight are restored, and the higher self-consciousness once more takes charge. The tendency is for the symptoms to pass off spontaneously, and to leave a residue of amnesia for the moment of impact.

Such then are the two extremes of concussion. In all but the very severe cases the reaction commences within a few minutes. If the restoration of consciousness is delayed for more than an hour or so, if the stage of reaction is prolonged for more than twenty-four hours, if stupor recurs after consciousness is regained, if there are signs of extracerebral damage, if there is local paralysis, or if blood is found in the cerebrospinal fluid, then it is clear that the substance of the brain has been grossly damaged, and that we are dealing with something more than the clinical state of concussion. None but the mildest cases escape some organic damage to the brain tissues; nor is the degree of concussion necessarily an index of the extent or the severity of the lesion.

*Cerebral contusion.*—What then are the possibilities as regards the underlying damage? By far the commonest are cerebral contusion, and laceration of the brain substance. Both cause focal necrosis, but the latter differs from the former because of the greater amount of hæmorrhage in laceration. In contusion the hæmorrhages are punctate; quite local in the milder cases, but irregularly dispersed and widespread in the deeply unconscious.

A usual thing at autopsy is to find the contusions localized at the poles of the brain. Sometimes the undersurface of a frontal lobe is severely bruised, it may be a temporal or occipital lobe, or there may be punctate hæmorrhages in the neighbourhood of a laceration. The area about the hypothalamus, with its sensitive vegetative centres, may be the site of these hæmorrhages, with resulting bradycardia, hyperthermia, and certain metabolic disturbances whose precise effect upon the tissues of the brain is still obscure. Again, these punctate hæmorrhages are found in the mid-brain or in the brain-stem. Another common feature at autopsy is a widespread pericellular œdema, a reaction phenomenon. Ritchie Russell [4] refers to a deeper condition within the substance of the brain, namely a fibrous glial reaction, and a reaction of the protoplasmic astrocytes. Greenfield [6], in describing speedily fatal cases, stresses the scattered distribution of the hæmorrhages, both in the white and the grey matter, and a local, rather than a general, distribution of the œdema. In cases coming to autopsy months or years later, he notes a degree of demyelination and scarring in the white matter; and if there has been a fracture, an extensive loss of cortical tissue. There is a general thinning of myelin sheaths rather than a complete loss of myelin; and to this fact, as well as to the scarring, he attributes the shrinkage of the brain substance.

Histological study shows changes in the nerve cells and fibres, changes in the walls of the vessels, and evidence of œdema. Axis cylinders are ruptured, astrocytes are swollen, and the microglia degenerates. Even minor lesions of the arterioles may damage their endothelial lining, and also affect the local contractile mechanism by which they respond to changes in internal pressure. Then there are the "ring" hæmorrhages due to blockage of the smaller vessels, and damage to the lining endothelium may lead to thrombosis. There may be fat embolism. All these evidences of histological damage are localized within the contused area, so that the damage is focal rather than general, and Greenfield believes that the thinning of the myelin sheaths is due to secondary œdema.

The contents of the skull differ in weight and in consistency; they differ, therefore, in their momentum. The brain substance has a certain elasticity, the blood circulating within the vessels of that substance is subject to ever-changing pressures, and the cerebrospinal fluid in the ventricles and subarachnoid space has a resistance of its own. The fluids have a potential power of yielding when the skull is suddenly distorted by the impact of a blow. The blood may be squeezed momentarily out of the cerebral circulation, and the cerebrospinal fluid may recede in part into the spinal subarachnoid space. The falx cerebri and tentorium resist sudden pressures and momentum, and the bony ridges on the floor of the skull and at the site of *contre coup* have a similar effect.

Much work has been done on the inco-ordination that results between the two cerebral circulations, the blood and the cerebrospinal fluid, in gross head injuries. This results from damage to the choroid plexuses and arachnoid villi. To this cause, as well as to the stretching of tissues, the dural septa, and serous membranes, are attributed the headaches and other distressing symptoms to be presently described.

Jefferson has stressed the point that the pressure of the cerebrospinal fluid is often low, certainly not notably increased; and that the effects of hypertonic saline are far from being uniformly successful in producing a return to consciousness. Moreover, operation under local anaesthesia not infrequently reveals a low-pressure brain. Hence the conception so commonly held, that compression is the cause of the stupor, is not entirely correct.

*Contre coup* would appear to be a question of the relative velocity of the skull, and the object with which it comes in contact. Thus, if a man falls on the pavement and hits his head, the brain may tear itself away from its coverings by its own momentum. In this way meningeal vessels are torn and bleed. But if a blunt instrument is applied to a man's head when the latter is stationary, it is probable that the resulting momentum of the brain substance causes it to impinge with great force at a point on the skull opposite to the point of application of the blow [9]. This causes hæmorrhage and laceration, often severe, at the former point, and results finally in dense scarring of the brain tissue in the damaged area.

To these pathological entities Jefferson [5] has added what he aptly terms the "epiphenomena"; that is to say extradural, subdural, and subarachnoid hæmorrhages due to laceration; together with the possibility of circumscribed collections of arachnoid fluid, a reactionary increase in the cerebrospinal fluid, and occasionally an internal hydrocephalus due to an obstructive arachnoid adhesion. Finally he lays great stress on the presence and clinical recognition of a delayed subdural hæmatoma.

Cerebral contusion is in its essence a traumatic encephalitis. If the patient has recovered full consciousness within twenty-four hours, we speak of it as an immediate or "minor" contusion; if the patient remains unconscious for a longer period, it is called a "major" contusion. This latter may precipitate an acute traumatic psychosis in which we see coma and stupor in varying degree. Whereas in concussion the classical features are widespread paralysis and coma, so in a major cerebral contusion its hallmarks are irritability and confusion. How much of the stupor is actually due to direct contusion, and how much to reactionary oedema? Patients are presented to us in varying depths of coma; they may be deeply unconscious, with widely dilated pupils, cyanosis, stertorous respiration and absent reflexes. In such cases death is speedy. Others are presented in a state of recovering consciousness; they can generally be roused sufficiently, dazed though they are, to co-operate in examination. Some relapse into unconsciousness; the crucial point is the depth of this unconsciousness. Light stupor and no physical

signs mean holding one's hand ; deep stupor and the presence of physical signs invite exploration. There are cases who are non-co-operative for hours, it may be for days ; in them the stupor varies in depth, but their general condition is not usually bad. They can scarcely be regarded as comatose ; they move about in the bed in a restless, irritable manner, and recover gradually.

Doubtless there is a large measure of reactionary œdema in these cases ; but there may be, for all the examiner knows, a focal hæmorrhage, and this may be accumulating at a varying speed. During the stage of reaction we see delirium at night, and in the daytime irritability, resentment, disorientation, and sometimes even violence. The patient lies curled up with his eyes away from the light ; if he complains of anything it will be of throbbing headache or photophobia. This stage of reaction reaches its height some two or three days after the injury. The semi-stupor and the full bounding pulse make one wonder how far this so-called " cerebral irritation " is due to pressure of some sort, either from hæmorrhage or œdema, and how far to inhibition of neural function. The condition nowadays is called " traumatic delirium ", which is a far better term than " cerebral irritation ". This stage will be prolonged in the presence of multiple contusions ; and the coma or stupor, as the case may be, will be modified if there happens to be a hæmorrhage elsewhere than in the head, for instance a coincident ruptured spleen.

*Recovery.*—The stages of recovery are subject to fluctuation and relapse. They always follow the same order, no matter whether the recovery of consciousness is a quick process, or whether it takes place, like a slow-motion cinema picture, in a series of clinical stages which can be recognized by the medical attendant. Some cases recover full consciousness in less than forty-eight hours ; others take days, weeks, or even months, even up to ninety days or more.

Broadly speaking, Symonds [10] perceives these stages under three headings : (1) Stupor, (2) Excitement, (3) Confusion. From flaccid coma the patient reaches a state of deep stupor, with no response to external stimuli except of the grossest character, alimentary and excretory. This deep stage of unconsciousness seldom lasts more than forty-eight hours, if as long. The next stage is that of excitement. The patient is dazed and bewildered, irritable, resistive, and resentful of interference. This stage may last for days or weeks, and is liable to setbacks for two or three days at a time. In the succeeding confusional stage the patient is quieter and more decorous, and his speech gains in coherence. Indeed, for a brief period he may speak quite rationally, and so deceive the observer. It will be noted, however, that he is still somewhat disorientated, that his memory is grossly impaired, that his judgment is faulty, and that he still has some disturbance of speech. On the affective side he is unduly elated, garrulous, and inordinately familiar with his attendants. He is quite unable to handle and co-ordinate the data governing a situation, no matter how simple ; he has lost the power of " synthesizing ", as Symonds puts it ; and lastly, he perseverates in action and in word. Whilst in this state of childish elation, he indulges in pseudo-memories and confabulation, just as one sees in the Korsakoff syndrome. During this confusional stage certain psychotic manifestations may come to the surface ; a paranoid state for example, a schizoid tendency, a manic-depressive tendency, or a change in personality. Finally there comes recovery of memory and insight. The patient may sit up suddenly and say " Where am I ? Whatever has happened ? " It becomes obvious to the observer at this point, not only that insight has returned, but that this is the time to test the degree of amnesia for the moment of injury, and the length of the retrograde amnesia. In the matter of amnesia one finds during the confusional stage that the blank interval appears longer than is actually the case. A recollection of facts which shortly preceded the accident is a sound indication that the patient has regained full consciousness.

*Progress.*—In assessing progress towards the return of consciousness, it is wise to note the development of speech. At first there may be nothing but a groan or a shout, constantly repeated. The vocabulary increases gradually, mainly as a repetition of words or a meaningless string of jargon. The patient pays little attention to questions. In the confusional stage which follows, he is still disorientated and garrulous; and his inconsequent flippancy is a gauge of the inhibition of his higher centres. His sense of the social decencies has not fully returned. Finally, with the return of orientation and a normal sense of inhibition, it becomes clear that he is beginning to reason and judge; at first he can deal only with single propositions based on a few data, but presently with matters which have a broader and more complicated basis. With recovery of insight, which is itself based largely on memory, speech falls into line, and the senseless perseveration of action disappears. Just as we are able on occasion, as for example in a subdural hæmatoma, to observe the descent from the rational state in series down to dementia, so in these cases we are privileged to observe the converse process from coma or stupor upwards in a series of stages to normal reason, insight, judgment, and control.

The temperature during the first twenty-four hours may rise from the subnormal state of the original concussion and shock to perhaps  $101^{\circ}$  F. In severe cases the pyrexia may last for more than forty-eight hours, the patient still being unconscious. In fatal cases the pulse is feeble and thready at the start, and its rate increases progressively until death takes place. On the other hand, if there is a healthy stage of reaction, the pulse, which was fast and feeble during the early stage of concussion, becomes slow and hard—a welcome sign. The respiration, at first rapid and shallow, deepens as recovery of consciousness advances. These three, the temperature, the pulse, and respirations, should all be normal within a few days.

There is no constant relationship between the degree of stupor and the pressure of the cerebrospinal fluid. The normal pressure is round about 160 mm. of water. Russell [4] found in his long series of cases that the pressure did not rise above the normal figure in three of the most deeply comatose cases. In seven stuporose cases it was below 200 mm., and in 12, where consciousness had already returned, it was above 200 mm. 330 mm. is about as high as the pressure does rise in these traumatic cases. Blood in the subarachnoid space is an irritant. It is very rapidly removed by phagocytosis, and a fall in the protein content takes place *pari passu* with the blood clearance. It may be that hæmolysis of free blood in this space and in the tissues is the cause of the temperature.

Throughout the progressive ascent from coma up to consciousness there may be setbacks. The patient may relapse into coma, a serious indication. These setbacks reveal themselves by certain danger signals, wisps-of-the-wind it is true in many cases, but enough in some to help us to decide whether the damage is due to cerebral contusion alone, or to something more than cerebral contusion. It is always difficult to convince lawyers and laymen that the brain may be damaged without injury to the skull, and indeed that damage to the skull is likely to absorb the main force of the blow, so that the brain itself escapes. It is equally difficult to convince certain medical men that there may be gross cerebral damage without physical signs. As a matter of fact, however, these physical signs are commoner than one might suppose. The difficulty is to elicit them because of the stupor and lack of co-operation on the patient's part. Thus, defects of smell and hearing, which are very valuable evidence, may not be tested for because the semiconscious patient does not complain of them. Pupillary signs are the commonest, eccentricity and reflex impairments (these suggesting contusion of the mid-brain), and grosser things like diplopia, defect of conjugate movement, and visual deficiencies. As a rule these disappear fairly rapidly. There may be epileptiform convulsions; and Parkinsonian features occasionally present themselves at a later stage. Incontinence of urine may continue, even after

consciousness has been recovered. The Hutchinsonian pupil, when present, is a matter of the first importance; and one may be able to detect a contralateral palsy even though the patient is in a state of stupor. To neglect physical examination of the nervous system just because the patient is unable to co-operate, would be a tremendous mistake. Indeed, the sooner an exhaustive examination of the nervous system and the cerebrospinal fluid is made the better. The data thus gained may be of inestimable value in comparison with those of examinations made at a later date.

*Residual symptoms.*—When the patient has recovered from the acute traumatic psychosis, that is to say from the confusional stages, we may profitably begin to analyse the residual symptoms of contusion, and to tease them out from any accompanying lesion. Trotter [3] taught us what these symptoms are: Headache, giddiness, irritability, nervousness, defective memory and power of concentration, fatigability, lack of initiative, and insomnia. To Trotter we owe the recognition of this combination of symptoms as an organic syndrome with an organic significance. Neurologists speak of the condition as “the post concussional syndrome”. It is not uncommon even at the present day for this group of symptoms in head injury without fracture to be looked on by unskilled observers as hysteria, or a compensation neurosis, or some other kind of psychogenic disorder. True, such neuroses may be superimposed in time on this syndrome of cerebral contusion; indeed they often are, as will presently be seen. The important thing, however, is to realize this, that in almost every case of cerebral contusion, no matter how minor it may be, there is some organic residue. If this fact is fully grasped, all the distressing sequelæ which are wont to follow in its train will be anticipated, and their proper measure taken if they occur.

The headache has its own very definite characteristics. It is a real pain, not the mere discomfort that the neurotic experiences, but a shooting, piercing, splitting, or throbbing headache; and it is induced or made worse by any change of posture such as bending down, and by coughing or sneezing. It is often complained of when the patient sits or lies in a definite position, when he retires to bed, when he wakes up, during physical exertion, in bright lights, and if the atmosphere is thundery.

The giddiness is a floating sense of unsteadiness rather than a true vertigo, with a feeling of rotation. It comes on when the patient stoops or gets out of bed suddenly; and transient though it is, it constitutes a source of perpetual inconvenience and discomfort to its owner.

Permanent mental downfall of a type that requires the care of others is unusual, unless the case is complicated by a massive hæmorrhage or a subdural hæmatoma; but minor degrees of mental disablement are quite common and may last for years.

*Subdural hæmatoma.*—It is not irrelevant at this juncture to discuss the possibility of the occurrence of a subdural hæmatoma; for this insidious and inscrutable sequel may come on acutely at an early stage, or weeks, or even months, after the original trauma. Its treacherous progress, or its sudden emergence in the guise of a rapidly increasing intracranial pressure, may well constitute an obscuring blur in the picture of contusion. Sometimes a large hæmatoma results from an insignificant polar contusion; again an extensively damaged cerebral surface may have a film of blood spread over it. It may occur in an acute form due to the tearing of cortical or pial vessels in the neighbourhood of the lesion, and be almost indistinguishable clinically from a case of extradural hæmorrhage. Sometimes there is a succession of hæmorrhages from a torn cortical vein, a tributary possibly of one of the dural sinuses.

This slow accumulation accounts for the latent interval. It may be that the lesion is bilateral in the region of the vertex.

Headache is the most constant symptom, frequently a unilateral headache. There may be vomiting and a slow pulse, and possibly papilloedema from increased intracranial pressure. The patient is drowsy and mentally confused, and the salient feature in the diagnosis is the fluctuation that occurs in the depth of the stupor. It may vary from a state of irritability in the less drowsy moments, down to a state of deep coma at its worst. There may be focal signs as well, a hemiparesis, a dilated pupil, or alterations in the deep reflexes. Convulsions may take place, cranial nerve palsies may present themselves, and the plantar responses may be extensor. If the lesion is bilateral, a hemiparesis may mislead one. The cerebrospinal fluid varies; it is frequently xanthochromic, on the other hand it may be quite clear. The case, when diagnosed, becomes one for operation as soon as may be; and surgeons are accustomed to explore both sides of the skull in the parietal region. Removal of the cystic mass yields an excellent result, if the intracranial pressure is not unduly disturbed in the process.

*Treatment.*—The routine treatment in cases of contusion is directed towards measures for dealing with the original collapse; for example, heat, raising the lower end of the bed, and strychnine if the respiratory centre appears to be embarrassed. The patient is placed in darkened surroundings. The lumbar puncture gives an indication as to intracranial pressure. If this is found to be high, prop the patient up; if low, keep him recumbent. If the pressure is near normal, then the head and shoulders should be moderately raised. If meningeal symptoms appear, as is usual in cases of subarachnoid hæmorrhage, then lumbar drainage will have to be undertaken. This must be done with caution, the manometer being brought into use; and no more fluid than is absolutely necessary must be withdrawn. Less risky, and equally effective from the point of view of reducing pressure, is the use of sucrose intravenously, and the employment of magnesium sulphate (30%) *per rectum* at six-hour intervals. At the same time one limits the intake of fluids. These methods of dehydration give the brain room to expand in its unyielding surroundings, and check the tendency towards a general spreading of reactionary oedema. The use of hypertonic saline is beginning to be called in question because it causes a reactionary rise. These and similar measures have superseded subtemporal decompression as a routine. Leriche recommended hypertonic saline intravenously in cases where the pressure was low. Chloral, the bromides, hyoscine, and luminal, all have their advocates, and make better sedatives than morphia which has a depressant action on the respiratory centre. Prolonged rest in bed and prolonged convalescence are inevitable in the severe cases; but in the milder cases this is not always a necessity. Simpson (11) has devised some interesting tests involving stooping and head-shaking to guide us as to the period of complete rest. It is most essential to avoid anything likely to lead to an anxiety state throughout the period of treatment.

Lambert Rogers remarks that since routine dehydration has been adopted right from the start in a large series of motor accidents at Cardiff, mental sequelæ, and indeed complications of all sorts, have become quite uncommon. "A striking change is the absence of cerebral irritation, and we no longer have cases shouting the wards down."

*Surgical treatment.*—Extensive scalp wounds and compound fractures of the vault require immediate surgical treatment, the removal of dirt and debris and fragments of bone being essential. Depressed fractures can as a rule be dealt with later. Subtemporal decompression may have to be considered in certain cases, where, despite routine dehydration, a progressive rise in pressure occurs. It is good practice to give luminal as a routine sedative for some months after convalescence is established.

*Delayed symptoms.*—If the patient is not rested sufficiently after apparent recovery, symptoms of what is known as "persistent contusion" may appear after his return to work. It is in this type of case that one is often left in doubt as to how much is organic, how much due to anxiety, and how much to impending legal or insurance proceedings. On the success of the lines of treatment indicated above and similar measures, the destiny of the patient largely depends; nor does one despair if there is a long period of stupor and confusion. The outlook of traumatic delirium and of persistent contusion are definitely hopeful in the long run, but there are still these residual symptoms and their sequelæ to be considered.

The headache of neurosis is a continuous discomfort rather than an agonizing pain. It arises from a painful memory; and inasmuch as amnesia for the moment of impact is an essential feature of concussion and contusion, this painful memory must be of a different order. Nevertheless, even though such a headache be discovered about this time, the scale should be weighed down on the side of organic injury, if there happens to be a history of stupor or unconsciousness lasting twenty-four hours or more after the original injury to the head.

Ritchie Russell [4] made a useful "follow-up" on the survivors of his cases. He examined 141 six months after their discharge from hospital; 55 had no abnormal symptoms, the remainder had residual symptoms. Headache was still severe in 13, and was more conspicuous in the young, and among those who had been unconscious for less than twenty-four hours. Dizziness persisted in 21; its incidence was greater as age advanced. Mental symptoms were chiefly of a minor degree; for instance a mild aphasia, difficulty in concentrating on one's business, deficiencies in memory, and indecision. Some of those who had been unconscious for more than twenty-four hours had already degenerated into a state of dementia. Nervousness was more common among the children; and in them changes of character, always for the worse, depravity, delinquency, and undue backwardness at school were more marked. Nine cases still complained of insomnia.

On the whole it may be said that the patients in our three Services will have been invalided before some of these residual symptoms have had time to appear; but in assessing their disabilities in terms of percentages, it is imperative that Medical Officers should realize very fully the organic nature of the lesion with which they are dealing, and the somewhat grim possibilities that may arise from it in the future. It is not merely a question of "fit" or "unfit" from a Service point of view. In the terms of their contract, enlisted men are entitled to treatment, and a full and clear knowledge of the sequelæ is inherent in the matter of sound treatment and in the thorny question of disposal of these unfortunate patients.

*Major sequelæ.*—Dealing with the insurance aspect of this subject, Trotter put the major sequelæ in the order of their importance as follows: Residual infections, epilepsy, cerebral neoplasms, and unresolved contusions. Residual infections are only common, however, after penetrating wounds, it may be years after the original damage. A secondary abscess results, due to foreign matter driven in with the original missile. This is the great danger of war wounds; hence the necessity of cleaning them up as early as possible, and extracting the bullet or splinter if it is accessible. Trotter [2] states that, "there is no evidence that injury has any effect in the production of a cerebral tumour". Epilepsy and the persistent cerebral contusion are the remaining considerations.

I am of opinion that this order must be modified from the Service point of view. For us it becomes a question of what can be recognized whilst the patient is still under our care, that is to say before he is invalided from the Service; and what may happen afterwards, not omitting from our calculations the possibility of

disciplinary proceedings, and compensation and insurance questions at any point in the course of the malady. I think the order should be as follows:—

- (1) Persistent cerebral contusion proper.
- (2) Such neuroses as may be imposed thereon.
- (3) Affective and emotional states which may develop.
- (4) Hysteria.
- (5) Epilepsy and its equivalents, with moral depravity and deterioration in personality, sometimes degenerating into a state of dementia.
- (6) Psychoses.

Two questions assert themselves persistently throughout the period of observation:—

(a) Can trauma be the actual cause of a psychosis, or is it merely a precipitating or aggravating factor?

(b) Can latent neurosyphilis be rendered active by trauma? These questions at once suggest others, for instance, what interval of time must elapse before the trauma and any subsequent mental aberration can be considered independent of one another, and how far do alcoholism or arterio-sclerosis modify the picture?

(1) Mapother [12] describes the residual symptoms of persistent contusion under the heading of "traumatic psychasthenia"; and it is well at the outset to underline his observations on the minor phases of the condition. For instance, it may not be apparent at a single interview that the patient is suffering from a collection of symptoms, which taken by themselves might be inappreciable, but taken together may quite reasonably destroy a man's usefulness for responsible work. Lack of initiative, retardation, loss of memory for engagements, and more especially indecision in a person accustomed to decide things, constitute the type of symptoms on which he lays stress. Again, there may be an inability to sustain continuous thought, and even a slight degree of confusion, so that the patient cannot think clearly.

(2) How often does one find such a series of symptoms dismissed as "traumatic neurasthenia", their organic origin being completely ignored? True, anxiety states may be superimposed on these distressing symptoms, and with good reason; for the patient finds himself wholly unable to adapt his mind to the normal routine of life, and particularly to the duties of his former occupation. He fears for his income, and compensation questions add to his burden. Some patients reveal intense anxiety lest they may be going insane. Wonted tasks, which they now find beyond their capacity to perform, render them irritable and depressed. The emotional state tends on the whole towards depression. It cannot be too strongly stressed that the persistent contusion has an organic origin; indeed it constitutes "a minor degree of that receding dementia which is a feature of the more obvious and prolonged mental disturbance" [10].

The consensus of authoritative opinion is that neuroses are not so very common after head injuries; what are so often registered as such are in reality the mental symptoms "of a major contusion, spread thin" [10]. Certainly, compensation neuroses and hysterical symptoms are no more common after head injuries than after injuries elsewhere. Nevertheless, mental stress may well be an aggravating factor in the genuine symptoms of contusion. "One can hardly conceive a greater mental strain," says Symonds, "than that of a lawsuit hanging over a man, who has nothing to show in evidence of disability besides his own word". There will always be some, of course, who make the most of their symptoms, in the hope of gaining thereby the maximum degree of compensation. These hysterically inclined persons are craving for sympathy, and rather than return to work, they take refuge in their symptoms. Then there are the actual constructive malingerers, who are more easy

to rebut. Purdon Martin declares, "We may scoff at these, but we must be careful to recognize that the difficulty of obtaining fair compensation gives rise, especially among the working classes, to a great deal of genuine anxiety" [13].

(3) Conduct, whether normal or pathological, is constantly tinged with emotional tones; moreover we are all creatures of instinct and habit. Behaviour is seldom based on a single conscious motive. It therefore behoves the Medical Officer who is dealing with the later stages of a head injury, to recognize and define the organic syndrome of contusion, and to estimate its intensity before proceeding to elaborate his diagnosis. Thereafter, he should contrast this estimate with whatever else may be imposed upon it, weighing all the facts first, and deducing motive afterwards. This duty he will best perform by going very carefully into the patient's previous history, his mental capacity and affective tendencies, before the occasion of the trauma, consulting relatives and fellow-workers, and comparing matter written before the injury and after it. It may even be worth while to obtain a series of photographs. Whilst performing this task he should recall that retarded intellectual processes and a defective memory, especially for recent events, form sound evidence of organic damage. In the neurasthenic proper, on the other hand, he will at once perceive the volubility, the wealth and the variety of the symptoms, and the typical headache, that is to say complaints of pressure on the vertex, or the feeling of a tight band compressing the head. Most important of all, the neurasthenic patient will recall and describe his accident, which in many cases was quite trivial, and reveal no amnesia for the moment of impact, which is the outstanding feature of concussion.

(4) Gross hysteria in these cases of accident is the result of pre-existing hysterical tendencies. These may be elicited by skilful questioning. The more markedly depressed cases are those who are constitutionally of a manic-depressive diathesis. If it be true, as is stated, that suicide is on the increase, particularly after motor accidents, it is possible that these naturally depressed cases are responsible for the rising figure.

(5) Epilepsy is wont to follow upon injuries to the head. Three questions at once arise. What percentage is so followed? How long does epilepsy take to reveal itself after the trauma? And, is this traumatic form of epilepsy a new feature in the case, or is it a latent idiopathic epilepsy brought to light by the injury? Trotter thinks that 5% of head injuries become epileptics in due course, and, that if epilepsy is about to occur, the tendency will have shown itself within twelve months. If after two or three fits the patient is free from attacks for two years, it is unlikely that he will become an habitual epileptic. Stevenson [14] puts the figure after war injuries at about 4½%. The first major convulsion is frequently preceded by attacks of *petit mal*, some of which come on at night, so that they pass unobserved. Then there are all sorts of epileptic equivalents, emotional outbursts, automatism, dream-states, paroxysmal headaches, and vertigo. Sometimes the disturbances are of a vasomotor character, for instance pallor, flushing, and sweating. More puzzling are the states of absent-mindedness, prolonged amnesia, and fugue. These sometimes alternate with true epileptic manifestations. Bolus [15], of the Ministry of Pensions, states that the longest claim between trauma and the first major epileptic attack was one of sixteen years, and that this claim was accepted by the Ministry.

Scar tissue and adhesions follow *contre coup* lesions and penetrating wounds. These drag, and sometimes result in epileptoid phenomena. Penfield and Foerster [16] have applied encephalography in these conditions, and they confirm the effects of traction which distort the ventricles. Wagstaffe [17] is inclined to

think that those who had Jacksonian fits just after the injury are more prone to develop epilepsy later on.

(6) Pronounced changes of personality and marked irritability, in a person usually stable, are usually signs of organic lesion [18]. Among these changes is one called "persistent demoralization". This is not necessarily associated with epilepsy. It is found most commonly as a sequel to head injuries in children, but was quite usual in adults for some time after the Great War. The condition may be defined as "habitual disregard, coupled with consciousness of the anti-social character of one's acts, in the pursuit of pleasure or the avoidance of displeasure" [12]. There is undoubtedly a psychogenic element in these cases, but they cannot be entirely explained in this way. Sometimes the disease is an isolated event, and sometimes it is associated with widespread deterioration. Parkinsonian syndromes may be accompanied by changes in personality; and another change worthy of note, is the epileptoid mental paroxysm. In the vernacular this is known as a "brain-storm". It is apt to occur in quite respectable characters after imbibing small quantities of alcohol. It may be purely post-traumatic, but is sometimes allied with schizoid or convulsive tendencies. The paroxysm is frequently accompanied by deeds of violence.

Most authorities are agreed that the incidence of epilepsy following head injuries after the Great War has been much underestimated; and Bolus is of opinion that simple through and through bullet wounds leave effects quite as severe as those of shrapnel and explosive shells. Moreover, epileptic dementia is more frequent and more severe in degree after gunshot wounds than in civil cases.

(a) The acute traumatic psychosis tends towards ultimate recovery. Major attacks of affective disorder, such as the manic-depressive psychoses, commonly reveal a neuropathic inheritance and a personal history. It would seem that trauma can precipitate a manic-depressive attack, or render an existing condition acute; but to connect the event with the trauma the psychosis must come on within a few months, and show some evidence of "bridging" symptoms. The schizoid or paranoid states which sometimes present themselves during the confusional stages of an acute traumatic psychosis, tend to clear up entirely. Korsakoff's syndrome also tends to disappear, and aphasia and agnosia and other cortical and subcortical symptoms gradually fade away. Should they reappear after a period of apparent normality, no matter how short, they are much more likely to be permanent [12]. Whether a trauma can cause, or even precipitate a schizophrenic psychosis, is a question which is rendered none the less easy to answer because of our lack of knowledge of the pathogenesis of schizoid states. That it can do so is the conclusion of Mapother, and his opinion seems to be gaining weight among psychiatrists; on the other hand, this is not in accordance with the statistics of the Ministry of Pensions.

The persistent contusion of trauma tends to ultimate recovery, but not necessarily complete recovery; there is sometimes a relapse with progressive deterioration down to dementia. This is more likely to occur in the presence of some toxic condition such as alcohol, or when arteriosclerosis or chronic disease complicates the picture.

(b) There remain to be discussed the connexion between trauma and neurosyphilis, and between trauma and alcohol, and finally the disposal of our cases. In the case of dementia paralytica there are no reliable statistics to guide us, and there are great variations in the rapidity with which the symptoms develop in non-traumatic patients. There is a general impression none the less that head injury plays some part, a "*locus minorus resistentiae*" for the spirochæte to lodge in. Kinnier Wilson was very sceptical about this; and demanded that, in order

to establish a connexion between the two, the symptoms should appear within forty-eight hours of the lesion. Other authors, however, consider that this interval may be as long as thirty months. One must not forget that the trauma may be the result of the disease, for in general paralysis injury to the head is quite a probable complication. As regards cerebral syphilis, it is well known that sudden delirium with paralysis of intracranial nerves often ushers in the active signs of the disease; but these are the identical things that occur in many cases after injury to the head. Hence the question is obscure. All that clinicians can say is this, that where latent disease exists, it appears to be rendered an active psychosis after trauma.

Alcoholics are liable to head injuries as a result of their drinking bouts; and it is a commonplace that injury to the head increases their tendency to suffer from the after-effects of quite small quantities of alcohol. The practical difficulty is this, that the symptoms of an alcoholic psychosis and those of certain post-traumatic psychoses are identical. We have already seen that a respectable imitation of the Korsakoff syndrome occurs during recovery from the post-traumatic acute psychosis. One thing, however, can be said with certainty about alcoholics, namely, that they tend to respond with psychotic symptoms to less severe head injuries than non-alcoholics, and that their prognosis is less hopeful in an injury of any given severity. It is sometimes difficult to decide whether a traumatic psychosis has been aggravated by the alcohol, or whether an alcoholic psychosis has been precipitated by the injury. If there was no known mental deterioration prior to the trauma, one would assume that the patient was suffering from a traumatic psychosis aggravated by alcohol. Sometimes a head injury precipitates an attack of delirium tremens, but the same applies to other surgical injuries where the head is not affected.

When invaliding the victims of injury to the head, we are confronted with questions of attributability or aggravation, and the degree of disability in terms of percentages. If the injury is attributable, then its sequelæ are obviously attributable too. The injury, however, may not be attributable; on the other hand it may have precipitated or aggravated a latent disability. In this connexion heredity and personal history are of vital importance, as well as the results of physical examination. The patient may have some organic disease, or be the victim of a toxic or degenerative agency, or of some metabolic disorder. Each case must be considered on its merits before "aggravation" is conceded. The organic syndrome of cerebral contusion should be much more fully recognized than is often the case, and it should always be remembered that the less obvious symptoms of persistent contusion, taken together, and elicited only after frequent examination of the patient and questioning of his associates, can render an apparently normal man quite unfit even for lighter and less responsible duties. If there is any retardation of intellectual processes, defect of memory, more especially recent memory, and particularly if the patient has lost the power of decision, the disability should be assessed at a much higher figure than is commonly the case.

The question of disposal is frequently a matter of anxiety to the Medical Officer in charge. One hesitates to send a man to duty if he has fits of depression, if his headache and dizziness are constant, and particularly if he is the victim of anxiety. Nothing makes these patients so irritable, and even anxious as regards their sanity, as to find that they are quite unequal to their accustomed labours. Should one decide to invalid, it is wise to warn the relatives of the man's partial incapacity, of the possibility of fits and their equivalents and emotional upsets, at the same time expressing hopefulness as to the outcome in the long run. In the case of a definite psychosis, one may be compelled to certify, but even in such a case a hopeful

outlook is on the whole permissible. The large bulk of the cases are not sufficiently mental to warrant certification. The Mental Treatment Act of 1930 can be invoked with advantage in many of them, and the relatives should be persuaded to cause the patient to become a voluntary inmate or outpatient at a mental hospital.

In assessing and appreciating the condition of these cases of residual mental trauma, it is well to bear in mind the clinical axiom that "It is the summation of small things that matters".

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## Section of Surgery

President—V. ZACHARY COPE, M.S.

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### The Treatment of War Fractures by the Closed Method

By J. TRUETA, M.D.

ADVANCE in the treatment of compound fractures has always been secured by the experience gained in time of war. Only in wars is there a sufficient number of these fractures to permit a study of all the problems involved. This was evident in the Great War, the last great conflagration which preceded the war in Spain. In the twenty years that have elapsed since the end of the Great War progress has, however, been maintained because of the increase of casualties due to road and industrial accidents. For this reason, the outbreak of the Spanish war found the study of injuries more advanced than at the end of the Great War.

Compound fractures in war may be produced by various projectiles: bullets fired from rifles or machine guns, artillery fire, and aerial bombs. Injuries from aerial bombs are always more destructive than others, even gunshot wounds, so that in this paper I shall give special attention to air raid injuries because of the gravity of the lesions.

Treatment of open fractures is not merely treatment of the bone but of the wound. Bone should be considered as organic tissue which reacts similarly to other tissues, so that the fracture is only a wound of the bone. Treatment of the wound is then the principal part of treatment of open fracture.

At all times the treatment of open fractures has been directed towards two objects:—

A. To avoid or to overcome local and generalized infection.

B. To obtain the best possible reduction of the fragments, and by immobilization to secure consolidation of the fracture without deformity.

In all surgical works of the past these two aims have been prominent. In the writings of Ambroise Paré, for example, his preoccupation with these problems is evident. Against infection he recommends a paste containing turpentine, comparable to the use of antiseptics three centuries later, and for immobilization of the fracture he advocates splints which are illustrated in his books. Paré was the first surgeon who opposed the custom of his time of cauterizing wounds by means of boiling oil with the idea of destroying the "poisons" produced in them. Like many other advances in surgery this innovation came about by accident. He described it in the following words:—

"At length my oil lacked and I was constrained to apply in its place a digestive made of yolks of eggs, oil of roses and turpentine. That night I could not sleep at my ease, fearing that by lack of cauterization I would find the wounded upon which I had not used the said oil dead from the poison. I raised myself very early to visit them, when beyond my hope I found those

to whom I had applied the digestive medicament feeling but little pain, their wounds neither swollen nor inflamed, and having slept through the night. The others to whom I had applied the boiling oil were feverish, with much pain and swelling about their wounds. Then I determined never again to burn thus so cruelly the poor wounded by arquebuses."

I will not waste further time with the history of the subject, but will discuss only the weapons we employ in the struggle against infection and deformity in open fractures.

#### *A. Prevention and Treatment of Local and Generalized Infection*

*Local antiseptics.*—From the time that Lister applied to surgery the discoveries of Pasteur, antiseptics were considered the most efficient means of avoiding infection of wounds. The great progress which Lister's technique represented was not, however, generally accepted for many years, and Lister had great difficulty in obtaining recognition for the new methods which were so opposed to the concepts in vogue in his time.

Beginning with Lister's carbolic acid and the mercury derivatives advocated by Koch, trial of new antiseptic products has continued up to our own day. Each new antiseptic has been an improvement on the one that has preceded it, both in its bactericidal power and its lessened toxicity for the tissues of the body. Progress continued until the time of the Great War, when Alexis Carrel introduced continuous irrigation with hypochlorite solutions. This was shown to be more efficient than any antiseptic used up to that time because it united to its bactericidal power a very weak toxicity for the cells of the body. The reason for the efficacy of continuous irrigation with Carrel-Dakin solution was not properly appreciated. The good results were ascribed to its great bactericidal power, whereas the real reason was that its slight toxicity allowed the tissues to react against infection. This was confirmed by Sir Almroth Wright, who obtained the same or even better results from the use of hypertonic solutions which had no antiseptic power but which acted physically in promoting drainage. Post-war experience has shown the doubtful value of antiseptics as a means of preventing infection of wounds, and in my opinion the war in Spain has confirmed this.

*Biological antimicrobial methods.*—The vogue for vaccines and serum, which some years ago was extensive, has also diminished. Apart from anti-tetanic serum and to a less extent anti-gangrene serum, the use of sera for all other wound infections fails to produce effective antibodies because of lack of specificity.

*Chemotherapy.*—Since the publication of the work of Domagk compounds of the sulphanilamide group have been widely used in the treatment of wound infections. This new method of treatment has been advocated with the same enthusiasm as was the introduction of the other forms of antiseptics. I shall not waste time discussing the underlying principle because it is well known. I am more interested to talk about our experience in its clinical application during the war in Spain. Apart from the complications which are observed when these drugs are used for severe infections and in large doses, such as nephritis, anæmia, erythematous rashes and petechiæ, I do not share the enthusiasm which was expressed in a monograph published in Spain during the war by Drs. D'Harcourt, Folch and Bofill (1938). I have not seen good results in severe infections of the limbs, though in pleuropulmonary infections and in some cases of meningo-encephalitis the results have been more encouraging. I believe it is necessary to use sulphanilamide compounds with prudence, never exceeding 6 or 7 grm. a day, and reducing it as soon as it is beginning to produce beneficial effects.

*Excision of the wound.*—Until the year 1897, when the German surgeon, P. L. Friedrich, published his experimental work on excision of contused wounds as if they were neoplasms, the factor of the vitality of the tissues as a decisive element

in the struggle against infection was not given its due importance. Since then, this principle has been appreciated, and in the Great War it was finally accepted. No treatment against infection, such as antiseptics of the wound or the use of sera, is employed to-day without first excising all damaged tissues, particularly those deprived of circulation. The only variation has been in the selection of the tissues which must be excised most radically. Contrary to the opinion held during the Great War by surgeons like Leriche, I believe that the greatest danger of infection lies not in bone but in muscle. Bone plays an almost passive rôle in infections and is only invaded secondarily after colonization of the organisms in the soft tissues. Dead muscle tissue is a favourable soil for the development of anaerobic infections, and for this reason must be excised meticulously. Small hæmatomata in the intermuscular spaces are good culture media which should be followed up systematically. Skin, on the other hand, provided it has a good blood supply, should be preserved, only the edges of the wound being resected with scissors. After the publication of Friedrich's work and the experience which followed it, but especially as a consequence of the Great War, a new factor gained importance, namely the time which elapses between the production of the wound and the removal of the devitalized tissues. As a result of the experience of the Great War eight hours was considered the limit of the optimum time for operation before infection of the wound occurred. But this limit is not exact because it varies in relation to the degree of destruction of the tissues. I have been able to see in hundreds of cases an aseptic course in wounded patients operated on after eight hours, provided the wound was but little contused, as for example in those produced by bullets fired from a distance. On the other hand, in wounds from shrapnel, and especially those produced by aerial bombs, infection occurs much earlier, often in less than four hours.

*Shock.*—Patients with compound fractures produced in air raids show signs of shock immediately after infliction of the wound, but this shock generally disappears with appropriate treatment, especially morphia, lobeline, heat, and transfusion of blood. Rest, physical and psychological, also helps to revive these patients. To this primary or psychic shock is added secondary or true shock when the wound has not been properly treated within a few hours. In extensive wounds of the limbs I have been able to observe clinically, perhaps for the first time on a large scale, that true shock is the result of absorption of disintegrating tissues. After air raids in Barcelona it has been possible to operate radically on hundreds of patients with severe wounds of the limbs within half an hour, and sometimes within twenty minutes. After treatment of the primary shock a meticulous operation was performed, all the devitalized tissues being removed and the limb itself preserved wherever possible. When operation was undertaken immediately, it was common to observe that secondary shock did not occur, nor did generalized infections develop. If true shock is to be avoided, I am convinced that operations for compound fractures with extensive wounds should be performed within the limit of two hours. In this I agree with Professor Manuel Bastos, who reached similar conclusions from experience in Madrid, where because of the proximity of the front it was possible to operate within this optimum period. Unfortunately, this limit of two hours cannot always be observed in the Army, because of the difficulty of transporting the wounded to the casualty clearing hospital, especially when the numbers are great and many have to be collected from between the two lines of fire. On the other hand, in bombarded cities the existence of civilian hospitals within the town allows immediate assistance to the injured, provided the organization is adequate.

*Physical methods. Open treatment.*—Among the procedures aiming at sterilization of the wound by physical means, open treatment has been most widely followed, and has given the best results. Suggested during the Great War by the German surgeon F. Schede (1915) and accepted immediately by Braun and later by Bohler, this method has produced excellent results. Schede used plaster casings

with windows as later did Bohler, while Braun only fixed the limb by a splint with the wound uncovered. The results that I have obtained by this technique have not been unfavourable, especially the promotion of healing under the slough, but it is of limited use in deep wounds of the limbs because retained secretions remain undrained under the slough. Only in severe trauma of the lower limb when there is doubt about the possibility of saving the limb is it necessary to watch the state of nutrition of the tissues for two or three days. In these cases it is advisable to employ open treatment, after immobilizing the limb correctly by continuous traction and a good splint such as Thomas or Braun. This treatment by exposure to the air is better than the use of antiseptics, because it does not interfere with the natural defence mechanism of the body, while allowing the surgeon to watch the wound without the necessity of handling it. Also it is painless and simple. This technique, like the use of antiseptics, is employed only after complete excision of the wound, i.e. after applying the principle of Friedrich.

*Drainage.*—One of the factors which favour the development of infection is retention of discharge, especially when, as in all war wounds, colonies of organisms occur in the depth of the wound. Good drainage is essential, for a badly drained cavity allows the collection of fluid, at first consisting of blood, but rapidly changing to pus. In my experience the best drain proved to be sterile absorbent gauze introduced between the tissues in the direction of the muscle fibres. Sometimes counter-drainage by a rubber tube was inserted through the plaster into the most dependent part of the cavity.

*Immobilization.*—It is well recognized that movement increases the dissemination of infection in the body and the absorption of toxins from wounds. This is probably due to interference with the local defence mechanisms through rupture of the small capillary and lymphatic thrombi by which the wound is isolated from the general circulation. Certainly it is true that a rigorous immobilization constitutes one of the most effective means of preventing and combating infection.

What do we understand by a rigorous immobilization? An immobilization which prevents movement of all the tissues but especially the most vascular, i.e. the muscles. This protection against movement can only be obtained by enclosing the extremity under a rigid casing which, while preventing all movement, even the most insignificant, permits a good circulation. The only known material which supplies this requisite is plaster of Paris. Experience in Catalonia is, on this point, convincing, as we shall see later. The only disadvantage of plaster is that it prevents examination of the wound at any given moment. Fortunately this examination is seldom necessary, but one must be quite dogmatic about the indications. In the upper limb, if the treatment has been correctly applied, progress is always satisfactory with the closed method, and gas gangrene and septicæmia are very rare. I have never seen these complications, even in patients treated by young surgeons with a limited experience. It is therefore not necessary to examine the progress of the wound. In the lower limb, thigh and leg must be considered separately. When operating on fractures of the leg, especially those involving the popliteal region or the calf, it is necessary to examine carefully both the circulation of the foot and the local circulation of the part, and especially the possibility of excising all devitalized tissues. When, in spite of this excision, there is still doubt about the vitality of the tissues that remain, or about the total circulation of the leg, it is essential to wait two or three days before putting on plaster, using instead open treatment with continuous traction which will permit frequent observation of the state of the circulation. In this way amputation can be undertaken without loss of time when it is apparent that the nutrition of the limb is insufficient. In the thigh the indications are similar, but with the difference that here the local circulation of the tissues surrounding the wound is more important than the general circulation of the leg, because the latter is more easy to assess. Here also if there is doubt one must wait two or three

days, keeping the patient in bed with the wound open, and the leg fixed by continuous traction. With this line of treatment it has been possible to save many limbs as well as lives, and to see gas gangrene disappear progressively from our hospitals.

I must consider here another question. Should these wounds be sutured or should they be left open? In this my criterion is absolutely definite. If suturing would produce the slightest tension in the soft tissues, the wound must be left completely open. If there is any doubt about the vitality of the tissues, especially the muscles, then here too the wound must not be sutured. It will be readily understood that in wounds from aerial bombs which constitute the greater part of my experience of war wounds, one seldom sees cases where suture of the skin is possible. On the other hand, in accidents of civil life, suture can, under certain conditions, be performed without danger.

#### B. *Reduction of Fractures and Immobilization without Deformity*

The only satisfactory method of obtaining reduction of fractures is by traction, initial or continuous. Reduction must be secured by the application of extension on the operating table. Each region has its own special technique. In the upper extremity it is possible in most cases to reduce the fracture by manual traction at the end of the operation. Occasionally in fractures of the humerus extension has to be made by transfixion with a Kirschner wire through the olecranon against counter-traction by means of a towel crossed around the thorax and fixed to the operating table. In fractures of the lower limb the deformity is reduced by traction, the details varying with the site of the fracture. To ensure proper reduction some form of orthopædic table should be used. The purpose of the traction is to reduce the fracture and maintain it in reduction so that after the surgical treatment of the wound is completed, a plaster cast can be applied.

Operation on the lower limb is performed with the fracture reduced by traction. In this way all the tissues are in their normal position so that both bone and soft tissues are more easily explored. Traction is made progressively in such a way that reduction is complete at the end of the operation. This is one of the reasons why X-ray examination is unnecessary before the operation. All the tissues with a bad circulation are excised, the bone fragments are placed in a good position, and the cavity drained with gauze.

*Immobilization with plaster of Paris.*—This should be put on while the patient is under the anæsthetic, profiting by the good reduction of the fragments that has been obtained by the continuous extension. *The patient should never be moved from the operating table in order to put on the plaster.* The reasons for this are clear: reduction of the deformity would be lost, time would be wasted, and shock increased.

*Plaster of Paris technique.*—To obtain complete immobilization both of the bone and the soft tissues, the plaster should be put directly on the skin without the interposition of cotton-wool or stockinette. Only the anterior superior iliac spine, the os calcis, and the tendo Achillis, should be covered by a thin layer of cotton-wool. In a few very thin patients it may be necessary to cover lightly the bony points around the elbow. The use of cotton-wool under the plaster diminishes the immobilization of the soft tissues and, because it quickly becomes saturated with the discharge of the wound, it increases the smell, so that the plaster cannot be kept on as long as is necessary.

*After-treatment.*—The plaster cast must be left in position as long as the smell is not excessive and the plaster does not become soft and wet. The patient nearly always runs a temperature for a few days after the operation, showing that a local fight against infection is going on at the site of the fracture. In a total of 1,073 patients treated in my clinic removal of the plaster for a true clinical infection was

necessary in only 0.75% of cases (*Lancet*, 1939 (i), 1452). On these occasions it was found either that a cavity was badly drained or that cellulitis had developed.

*Advantages and drawbacks.*—The immediate benefits of the plaster treatment are absence of pain, rapid disappearance of shock, elimination of sleepless nights, and return of appetite. The absolute rest of the fractured limb promotes good normal union. The failures can all be attributed to faulty technique; the surgeon had either tried to save a limb with insufficient blood supply, or had not excised enough bruised tissue.

The most common complications are cellulitis and lymphangitis. Diagnosis and treatment are usually easy. If in a case of fracture treated by the closed method there is a rise of temperature, loss of appetite, pain at the site of fracture, sensation of tension and regional adenitis, the plaster must be removed. Nearly always cellulitis or more rarely a primary lymphangitis is found. The usual treatment for cellulitis is carried out. The intermuscular planes are widely opened up, the whole of the affected area is exposed, and the cavity drained with sterile absorbent gauze. The plaster is renewed either immediately or at most two or three days later. I have had to remove the plaster from 8 of my 1,073 patients for this reason.

*Gas gangrene.*—In the case which is developing gas gangrene the symptoms are immediately more striking. The patient says he cannot sleep, he has no appetite, his tongue is dry, he is restless, and from the first moment his pulse-rate is raised. Locally there is increasing pain in the region of the wound, local heat and tension. The foot or the hand becomes rapidly cold, in a few cases with oedema, and the digits cannot move. With this combination of general and local symptoms, the plaster should at once be removed and the wound opened up without waiting for evidence of gas beneath the skin. Bold resection of the skin and devascularized tissues with separation of the intermuscular spaces followed by immobilization has been successful in several cases transferred to my clinic for treatment.

This complication, which in my patients occurred once in 1,073 cases, generally develops within the first two or three days after the infliction of the wound. For this reason it is essential to be extremely vigilant during this time and not to encase in plaster fractures where there is doubt about the vitality of the tissues. In open fractures treated by other techniques with the object of watching for the appearance of gas gangrene it is important also to diagnose the condition before bubbles of gas are seen, relying on the general state and the local signs. No time is lost then if vigilance is sufficient in the cases treated by the closed method, and as I have said before, if the technique is correct the complication is rare. In the majority of cases of gas gangrene that I have seen, either the limb has been preserved in the presence of vascular lesions that interfered with the normal circulation, or the wound had been sutured.

*Plaster windows.*—I am convinced that making a window in a plaster case neutralizes all the value of the plaster treatment. Such an opening interferes with the local circulation at the site of fracture, sometimes producing great oedema, disturbs the immobilization of the soft tissues and, if the opening is large, the immobilization of the bone fragments. Those cases of compound fractures which cannot be closed under plaster because of anxiety about the vitality of the tissues are best treated by continuous extension with the Thomas or Braun splint.

*Special benefits of the closed method for the treatment of war fractures.*—The widespread use of plaster of Paris in Catalonia was brought about by the exigencies of war, but the reasons which compelled me to advocate this method were the following:—

- (1) It is the best complement to the technique of local excision of the wound as a means of avoiding infection.
- (2) It allows immediate radical operation of fractures, because after the operation the patient can be moved without danger and evacuated long distances.

(3) It permits a perfect drainage of the cavities of the wound because the plaster acts by suction of the discharges absorbed by the gauze. For this reason I do not use vaseline, which diminishes absorption.

(4) It avoids the dehydration and loss of heat that occur in large wounds if the open method is used and which induce a condition of shock.

(5) If the plaster is correctly applied, that is, directly on the skin without the interposition of cotton-wool and with the limb in correct position, displacement of the fragments does not occur. Frequently the first plaster achieves the desired position, and at each change of plaster the fragments are maintained in the position that was obtained on the operation table. In other cases a better reduction must be secured after ten or twelve days when the danger of severe infection has passed, because the need for speed at the original operation made it impossible to place the fragments in a sufficiently good position. This can be done (with X-ray control) at the base hospital far from the danger zone and without urgency.

*Confirmation of the value of this technique.*—In the first place I must mention our results in Spain. Mine have been published in my book on the treatment of war wounds and shortly Colonel D'Harcourt, chief surgical consultant to the Republican Army, will publish in an English journal the results obtained in 5,000 cases treated under his direction in the Military Hospital in Barcelona. As I have mentioned earlier, I had one case of gas gangrene in over 1,000 cases of compound fractures, and Colonel D'Harcourt had 20 in 5,000. It is necessary to take into account the fact that I was able to operate immediately on many of the patients, and that in others I was the first surgeon who treated them. Dr. D'Harcourt, on the contrary, was obliged to treat many wounded soldiers admitted to the military hospital in a bad condition after having been inadequately treated previously or treated on wrong lines. In view of the small number of cases of gas gangrene in the recent statistics of the Catalan hospitals there has grown up a doubt as to whether, in our country, gas gangrene organisms exist in the same proportion as in other countries of Europe. On this point I can affirm, though I have no statistics available, that the number of cases of gas gangrene in our war was extraordinarily high, but they occurred only in places where proper treatment was not used. It is only necessary to glance at the *Revista de Sanidad de Guerra* to see the cases reported there. For example, in an article by Dr. V. Goyanes Alvarez (1938) on transfusion of blood in two hospitals in Madrid, 61 transfusions were given for gas gangrene. In Madrid also there was a hospital devoted entirely to the treatment of this condition. I have information that gas gangrene was a frequent complication among the wounded of General Franco's army, and appeals were made to certain countries to send quantities of anti-gangrene serum for treatment. And both the Republican Army and the Army of General Franco fought on the same soil and under the same climate. I maintain, therefore, that if in Catalonia gas gangrene disappeared, this was due to the technique which our surgeons employed.

*Opinion of foreign surgeons.*—As is well known, at the end of the war in Catalonia, a retreat from the country of nearly half a million persons took place. Amongst these refugees who went to France were over 10,000 wounded, nearly all with wounds of the extremities. Numbers were evacuated in army ambulances but many thousands went across the frontier walking with plasters that covered and immobilized open fractures. Several hospitals were organized in France attended by surgeons of "La Santé Militaire Française" who did all they could to attend to this unprecedented influx of wounded. During the early days of the retreat the French doctors were amazed by the arrival of thousands of soldiers—many on foot—in bad general condition due to fatigue and hunger, with plasters smelling offensively, under which were extensive wounds. Many amputations of urgency were performed because the surgeons feared the development of gas gangrene.

A short time later reports were presented to medical societies in different parts of

France on the observations made by these surgeons, who had previously had little experience of the closed method of treating compound fractures. The general consensus of opinion was that the results were eminently satisfactory, serious infections being extremely rare. (*Soc. de Chir. de Toulouse*, Feb. 1939, *Soc. de Chir. de Lyon*, May 1939.)

Dr. M. Arnaud and others reported to the Academy of Surgery in Paris (May 1939) the results they had seen in 800 wounded from Catalonia, the majority of whom had been treated by the closed method. They were surprised to see only one case of gas gangrene and were impressed by the favourable progress of the wounds and the absence of general infection.

All the wounded who came to France during the evacuation of Catalonia arrived after many days of travelling from one place to another under the psychological and material conditions of an army in full defeat—conditions which in every war produce the highest mortality. In face of this the testimony of these French surgeons is an additional proof of the value of this method.

I hope that this evidence will be sufficient to encourage my British colleagues, and I am sure that the result to life and limb of treatment of compound fractures in the present war will be very different from that of the Great War.

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*Discussion.*—The PRESIDENT said that the contribution which Dr. Trueta had just given to the Section was a momentous one. It bade fair to revolutionize one of the most difficult problems which faced surgery, especially in war. He hoped many members would be willing to discuss some of the problems he had brought forward.

Mr. A. D. WALL said that he had recently arrived back from China. In 1937, in the war which then enveloped Shanghai, he had tried out the plaster immobilization method which Dr. Trueta had used in Spain and got exactly similar results. He could confirm everything that Dr. Trueta had said about that method. With regard to the contention that the method might not be successful on the soil of France, he could say that there was no more heavily manured soil than that of the Yangtze delta on which Shanghai stood. Although not much gas gangrene was seen in the hostilities around Shanghai, they had had experience of gas gangrene, especially in what he might call the gunshot wounds of civil practice, for in Shanghai there was not infrequently a "war" between police and gangsters in which wounds were sustained, and even prior to the outbreak of the Sino-Japanese hostilities he had been accustomed to treat many gunshot wounds of this nature.

Dr. LEONARD COLEBROOK, speaking as a bacteriologist, said he had spent a long time in the last war working on wound infections, and his interest in the subject had continued ever since. In considering Dr. Trueta's most interesting work he could not help wondering whether one of the essential pieces of evidence had not been omitted. At all events he had been disappointed not to get one such essential piece of evidence, namely, as to whether hæmolytic streptococci were actually present in these wounds that were encased in plaster. There had been a great deal of concentration upon gas gangrene, but he took it that in the Great War the hæmolytic streptococcus was a far more important enemy than the organism of gas gangrene. He thought it was true to say, although not generally recognized, that 80 or 90% of all the open wounds in the British base hospitals in the last war were infected by hæmolytic streptococci, and an enormous number of deaths were due to that organism rather than to the gas gangrene microbe. It would therefore be of great interest to know whether these organisms were present in the Spanish cases which had been treated under plaster. He thought that it had been learned in the last war that

if the hæmolytic streptococcus was not present the wounds could take care of themselves, whether they were sutured or not. If, on the other hand, the hæmolytic streptococcus was present many troubles occurred, such as cellulitis and septicæmia, and not infrequently death resulted.

He asked if there had been no deaths among the 1,073 cases which Dr. Trueta mentioned. He understood there had been eight cases of cellulitis and one case of gangrene.

Dr. TRUETA replied that he had six deaths. One from shock and one from pyæmia. One patient died from gas gangrene and another from wet gangrene. The remaining deaths were due to bronchopneumonia and a pulmonary embolus.

Dr. COLEBROOK said he had gathered from Dr. Trueta's book that when failures occurred they were due to deficient drainage. He wondered whether these were actually cases in which there were hæmolytic streptococci. There seemed to have been little pathological investigation carried out during the Spanish war.

Dr. TRUETA replied that all types of streptococci and staphylococci had been encountered in the cases dealt with in his clinic. There was not one case without such organisms, and he believed it was only the treatment which stopped absorption and prevented severe infections. Hæmolytic streptococci were frequently found in the wounds without generalized infection or other complications. When the plaster was changed there was always a slight rise of temperature and some local pain, lasting only for one or two days. He believed that the immobilization of the soft tissues was the reason why complications were so few.

Dr. C. A. R. SCHULENBURG said that Dr. Trueta had mentioned as one of the drawbacks in dealing with these cases the horrible smell caused by the saturation of the plaster with the discharge of the wound. This might to some seem a small disadvantage, but it must be remembered that while the doctor saw the patient for only a few moments, his fellow patients and the nursing staff were continually in his proximity, and, having to endure the smell night and day, became rebellious, so that one might be forced to change the plaster prematurely. He wondered whether Dr. Trueta had tried any local measures of combating the smell and if so with what result.

Dr. TRUETA replied that in some cases brewer's yeast was employed as recommended by Professor Leriche, and in these the smell had not been so pronounced. A certain smell remained but with this technique it was only moderate because the local fermentation of the yeast absorbed the products of the disintegration of the tissues and the result was a cleaner wound. This technique did not interfere with the excellence of the result in other respects. He had employed it in only a few cases, however, because the problem in the later part of the war was to obtain the yeast. Where the yeast was employed there was undoubtedly improvement, but the best prevention of excessive smell was early operation before infection of the wound occurred.

Mr. A. TUDOR EDWARDS asked whether the yeast was put on the plaster or actually in the wound.

Dr. TRUETA replied that it was put in the wound.

Mr. G. GORDON-TAYLOR paid a tribute to Dr. Trueta's paper, to which they had all listened with intense admiration and interest. There was much in what he had brought before them that was novel and might almost be said to be iconoclastic. But all surgeons worthy of the name preserved the open mind, and methods of surgery changed. In any case the illustrations they had seen that day were eloquent proof of the efficacy of the methods which Dr. Trueta had championed, and it might be that in the days to come these methods of excision and immobilization in plaster would prove to have been the method of election in the war of 1939 in which the British Empire was engaged.

Mr. W. C. GISSANE said that he had followed this treatment in dealing with civilian injuries. On changing the plaster in those patients who had sustained gross muscular and bone injury it was extremely difficult to hold the original reduction of the fracture. If it was held by traction he found that the fragments became distracted. He desired to know if Dr. Trueta had found any method of overcoming that great difficulty.

Dr. TRUETA said in reply that the necessity for changing the plaster depended on the bad smell. If there were any way of stopping the smell it should be possible to retain the first plaster to the end. In the absence of clinical necessity for a change, the first plaster was retained longer than the others, generally for about six weeks. The smell was very disagreeable. In most injuries from aerial bombs there was considerable loss of soft tissue; they were not entirely analogous to those of road accidents occurring in civil life. When changing the plaster in fractures of the upper limb the fragments were easily maintained in their proper position and no traction was necessary. In the lower limb because of the loss of tonicity of the muscles, it was necessary in a minority of cases to maintain the fragments in position by means of a Kirschner wire or a pin while the second plaster was being applied. Sometimes, as he had stated in the paper, it was desirable to change the plaster after ten or twelve days in order to obtain a better reduction than could be secured at first, in view of the danger of severe infection. This better reduction must always be obtained under X-ray control.

In reply to a further question whether, in between the first and second treatments, when extension was being made, he took out the pin immediately, he said that when the plaster was dry the pin was taken out at once.

Lt.-Col. D. C. MONRO asked whether Dr. Trueta could give any idea as to the amount of plaster which one surgical team would use in an average day, or the weight of plaster used per day.

Dr. TRUETA (in reply), said that when the press of work was great a special plaster team prepared the plaster while the operation was being completed and applied it as soon as the operation was ended, while the surgeon went on to the next case. It was not possible to describe an average day. For example, on March 17, 1936, at his hospital, 200 plaster cases were dealt with in twenty-four hours. On a busy day as much as 600 or 700 lb. of plaster might be used.

The PRESIDENT, in closing the proceedings, said that one point in Dr. Trueta's remarks linked itself up in his mind with Lord Lister. Dr. Trueta had said that from four to eight hours was about the limit of time which might be allowed to elapse between the production of the wound and operation before infection of the wound occurred. In Lister's original paper on the treatment of compound open wounds and fractures, he stated that if after six hours the antiseptic treatment were applied it would not be certain in its effect, whereas when the application was made before six hours had elapsed the results were uniformly successful.

## Section of Comparative Medicine

President—C. H. ANDREWES, M.D., F.R.S.

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### Latent Virus Infections and Their Possible Relevance to the Cancer Problem

#### PRESIDENT'S ADDRESS

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ANIMALS and plants may harbour viruses within their cells, though showing no evidence of disease; bacteria, too, may carry a bacteriophage which does not obviously affect them. Such phenomena may be called "latent virus infections".

#### VARIETIES OF LATENT VIRUS INFECTIONS

Virus infections which do not obviously affect the host may not all have the same natural history. Let us consider the several varieties; we shall note that they are not sharply separable from one another.

##### (i) *Transient Latent Infections*

Under this heading we include those diseases in which an animal becomes infected with a parasite, the infection runs its course and the parasite ordinarily ceases after a time to be recognizable; at no stage, however, do overt symptoms of disease occur. We shall see later that a small quantity of residual virus may be very hard to demonstrate in a recovered animal, and non-recognition of a virus may thus be due to the imperfections of our technique. It would be more accurate, therefore, to call our first group "Apparently transient latent infections".

The natural subclinical immunization of many human beings against diphtheria may be ascribed to transient latent infection, and there are many examples to be found amongst virus diseases. For example, yellow fever occurs in a wide area of tropical America and Africa and convalescents from the disease have in their sera neutralizing antibodies active against the virus. But many of the inhabitants of these regions have antibodies in their sera though they have never suffered from classical yellow fever. Some may have undergone an attack of yellow fever manifesting itself as an influenza-like infection and of these 90% have yellow fever antibodies, but of those who do not even give this history, 47% have specifically neutralizing sera (Soper and de Andrade, 1933). There is no dispute that yellow fever antibodies occur only as a specific reaction to infection with the virus, and it thus appears that yellow fever can exist as a symptomless infection.

During the course of influenza epidemics, sera of many persons in the epidemic areas show substantial rises in neutralizing antibodies against the virus though the people concerned have remained in perfect health throughout the epidemic (Stuart-Harris, Andrewes and Smith, 1938; Francis, *et al.*, 1937). The majority of human adults, too—80% in American cities—have antibodies against poliomyelitis virus in their blood: their significance is disputed more than that of the antibodies active against yellow fever and influenza, but most workers agree that they probably indicate past contact with, and often inapparent infection with, the virus of infantile paralysis. Amongst diseases of birds we may notice psittacosis. According to Meyer and Eddie (1934), a normal course of events is for budgerigar nestlings in aviaries in California to develop a symptomless infection with psittacosis virus; this is

associated with enlargement of the spleen from which virus is usually not recoverable after the sixth or eighth month of life.

In the examples just quoted the infecting agent is capable of producing, according to circumstances, a clinical or a subclinical infection in the species attacked. Nicolle and Lebailly (1919) used the term "infection inapparente" to denote particularly an infection which in a particular species was always symptomless. Thus, the *Rickettsiæ* of typhus fever which they studied produced no symptoms in the rat, could be carried on indefinitely by rat-to-rat passage without the production of symptoms but would cause symptoms again on inoculation back to the guinea-pig. Burnet (1936) has described the reaction of the rat to louping-ill virus. In this instance, virus introduced up the nose of a rat will pass to the olfactory bulbs but as a rule no further; no symptoms are produced and continued passage in series is not possible. This would seem to be a true instance of "infection inapparente" in Nicolle's sense, but Burnet and most English writers use the term inapparent infection as synonymous with latent infection. The distinction is not clearly defined and it seems better, therefore, not to treat "infections inapparentes" as a separate category of latent infection.

#### (ii) *Persisting Virus Infections*

After an overt attack of a virus disease, the responsible virus may continue to be harboured by a host at a time when the disease-symptoms have long disappeared. Poliomyelitis virus has been recovered from nasal washings of man during convalescence, and the very similar virus of mouse-paralysis (Theiler, 1934) has been found in the spinal cords of recovered mice for as long as a year after infection. Fowls infected with the virus of fowl-pox continue to carry the virus for long periods, particularly in the liver, and after infection with laryngo-tracheitis (Gibbs, 1933), the virus of that disease may be found in the trachea of fowls for as long as two years. Since most animals infected with a virus develop antibodies against that virus in the course of their disease, it may be a hard task to demonstrate a small quantity of persisting virus in the tissues of a recovered animal, since it may be neutralized by the antibodies present when the tissues to be tested are ground up and extracted. Special techniques have been devised for overcoming this difficulty; Olitsky separated virus from the antibody in recovered animals by cataphoresis and thus revealed the presence of vaccinia virus in the tissues of convalescent rabbits for as long as 133 days after infection. The virus of foot and mouth disease was detected in the urine of hyperimmunized cattle 246 days after infection (Waldmann, Trautwein, and Pyl, 1931) by a method involving adsorption of virus on to charcoal; here, however, they were probably dealing with concentration of virus and not solely with separation from antibody.

In some instances potent antibodies are not recognizable in the recovered animal and then virus may be revealed more easily. Traub (1938) could as a rule find no antibodies in the sera of mice recovered from lymphocytic choriomeningitis infection and he had no difficulty in showing that virus was present for long periods in many such animals. In the case of psittacosis-infected mice, Bedson (1938) was able to find virus in the spleen for seven months; psittacosis is another disease in which potent neutralizing antibodies are hard to demonstrate. A classical example of persistent virus infection is equine infectious anaemia. A horse may be attacked by this virus and may show comparatively trifling symptoms or none at all over a period of years; and yet a few drops of its blood taken as long as fourteen years after the original infection and injected into another horse have been known to produce a fatal infection; here, too, antibody production is not in evidence (Schalk and Roderik, 1923).

Many writers on viruses have sought to explain the long-continued immunity which follows many virus infections as being an infection-immunity, an immunity which only persists because the virus has never completely died out of the body. Of recent workers Webster (1938) suggests that the immunity of mice

vaccinated against St. Louis encephalitis virus may only endure while virus is still present, particularly in the spleen, while Bedson (1938) has made similar suggestions concerning the immunity of mice to psittacosis. Where virus can be shown to persist for a long time it is easy to believe that such persistence is responsible for the long immunity. Even where no virus can be recognized, its presence might very well be suspected, though masked by co-existing antibody. It is very possible that persistence of virus is the rule and yet we can only recognize that persistence is only possible when neutralizing antibodies cannot act to hide the virus. We must, however, be cautious and not claim that persisting virus is always the cause of long-enduring immunity until we are surer of our ground.

(iii) "*Indigenous*" Viruses

Interesting as are transient latent infections, which are apparently self-limited, and the instances of viruses persisting after overt infections, a third group is perhaps more significant still. These may be called "*indigenous viruses*". The group includes those viruses which in their behaviour combine the properties of the first two groups: as in our first group they cause symptomless infections, which, however, are not self-limited; as in the second group the viruses persist indefinitely in the host. There is a third characteristic of "*indigenous viruses*": they commonly infect their host very early in its life. They may make their entry in infancy or childhood, or *in utero* or may even be handed down from the parent in the germ-plasm. Thus an animal or plant may be born with a virus-infection of which the presence can only be recognized indirectly. One may by introducing some stimulus, upset the virus-host equilibrium, and cause the appearance of obvious disease or one may transfer the infection to a susceptible individual, an "*indicator host*". (This term will be discussed later.) This type of host-parasite relationship is what Theobald Smith (1934) has considered as perfect parasitism, an association in which the two partners are perfectly adapted to one another, neither causing the other any inconvenience and with admirable arrangements for perpetuating the partnership from one generation to the next. Of this nature are the symbionts which are found in the cells of many insects: they are believed on morphological grounds to be usually bacterial in nature or perhaps related to Rickettsiae, and some at least have been cultivated on artificial media (Glaser, 1930). They may be transmitted "*through the egg*", but the transmission is rather "*on the egg*" than actually through the germ-plasm; eggs are contaminated by the symbiotic agents which are present in the ovary at some time before the eggs are laid.

*Lysogenic Bacteria*

There are now overwhelming grounds for believing that the bacteriophages are of the same nature as viruses, are in fact viruses which parasitize bacteria. It is also established that many bacteria, so-called lysogenic bacteria, regularly yield filtrates containing a phage, active, not upon the strain "*carrying*" them, but upon some other sensitive strain. Such a strain can be used to test for the presence of phage and is therefore called an indicator strain. Many strains of *B. coli* for instance carry phages which are active on Shiga dysentery bacilli (Lisbonne and Carrère, 1922); *B. sanguinarum*, again, is a very valuable indicator organism for lysogenic *Salmonellas* (Burnet, 1932). For some time it was supposed that diphtheria-bacilli were not subject to phage-action; then Smith and Jordan (1931) showed that all diphtheria bacilli were lysogenic, carrying phages active against an indicator strain of *C. diphtheriae*. The diphtheria phages had not been detected before simply because no indicator strain had been discovered.

Burnet (1932) showed that nearly all *Salmonellas* were lysogenic, carrying one or more of several different phages. He has pointed out that the multiplication of phage and bacterium must be exactly co-ordinated so that each daughter-cell receives some phage when cell division occurs. Lysogenic bacteria afford an excellent example of the indigenous virus, an example, moreover, in which the virus is handed down in the germ-plasm—in so far as we can use the term germ-plasm with respect to a

unicellular organism. Why it is that a lysogenic bacterium is not lysed by the phage it carries we do not know. Does it lack some structural element which the phage must attack before it can dissolve the bacterial wall? Is there some little understood mechanism for intracellular restraint of the parasite? And if so does this act by preventing the phage from building up its concentration to the threshold for lysis? An answer to these questions might prove of the utmost importance in the virus field generally.

It seems clear in any case that the phage-bacterium equilibrium is not necessarily wholly stable. Cultures of certain lysogenic bacteria may exhibit "nibbling" of the colony edges from time to time, a phenomenon suggesting that the bacteria, or at any rate some variants occurring in the culture, are not wholly insusceptible to the phage which is being carried. Curious appearances which turn up in cultures of *B. pyocyaneus* can also be best explained in terms of incomplete resistance on the part of lysogenic organisms. Lominski (1938) has described bacterium-phage associations of varying sorts: one may find frank lysis of a culture, and lysogenic strains. But besides these are attenuated lysogenic strains, strains in which the only demonstrable effect of the phage which is carried is to render the bacterium resistant to the action of a related but more virulent phage. Or again in a "crypto-lysogenic" strain the lysogenic character of the culture may be wholly hidden unless one alters the environment by making the culture grow under difficulties, when evidence that a phage is being carried may reappear.

We shall discuss later how such upsets of a host-parasite equilibrium may be important in explaining phenomena of disease in animals.

#### *Indigenous Plant-viruses*

It has been shown by Johnson (1925) that almost all strains of potato under cultivation in America "carry" a virus, for extracts of American potato plants inoculated into tobacco and certain other solanaceous plants will regularly produce a disease having the properties of a virus infection. In fact it is possible that "normal" potatoes may carry more than one virus. Such virus commonly produces no symptoms in potatoes; but after several passages through tobacco its virulence may become exalted until it can cause disease when inoculated back to its original host. In calling this an indigenous virus we must bear in mind that vegetative reproduction is the rule in the artificial propagation of potatoes and that potatoes raised from true seed do not apparently carry the virus from their youngest stages. Latent plant virus infections exhibit one important feature: they may render the host-plant refractory to infection with a related but more virulent strain of virus (Salaman, 1933).

There is some evidence that with higher plants, as with bacteria, change in environment may upset the virus-host balance; thus the viruses of crinkle and potato mosaic produce symptomless infections when grown at over 20° C. but characteristic symptoms when the temperature is reduced below that level. In other instances abnormal cooling masks symptoms, as when tobacco plants infected with mosaic are grown at temperatures below 7° C. (Bawden, 1939).

#### *Indigenous Viruses of Animals*

Most animals differ from plants and bacteria in their ability to form antibodies, and we have already seen how viruses persisting after an attack of disease may remain masked by co-existing antibody. It might therefore be expected that the presence of indigenous viruses would be harder to detect in the animal than in the vegetable kingdom. One or two interesting examples are, however, known to us.

(a) *Lymphocytic choriomeningitis in mice*.—It is doubtful whether the virus of this disease is a mouse virus which occasionally infects man or a human virus which has managed to establish itself in mice. The equilibrium established between this parasite and a colony of mice has been described in some very important work by Traub (1939). In his earlier studies he noted that virus persisted in the blood of

mice after recovery from infection and that female mice thus carrying virus could infect their young *in utero*. Such young mice showed symptoms of disease, particularly tremors and inco-ordination. Other young mice not infected *in utero* became infected by contact soon after birth and showed no definite symptoms, only a decreased growth-rate. In such mice it was possible at a certain stage of the infection to produce frank symptoms by the intracerebral inoculation of sterile broth—another result of disturbing a host-virus equilibrium. Dr. G. M. Findlay tells me that one stock of mice in England carries choriomeningitis virus and it is possible to provoke symptoms in them with some regularity by intracerebral broth-injection. Let us return to Traub's mice: as virus and mice lived together for two years the state of affairs gradually changed. Infection of young mice *in utero* became the invariable rule, apparently because all stock mice, young and old, were now carriers. Further, the disease had gradually become milder till it produced no symptoms at all; all mice carried virus from birth and had an infection-immunity throughout life. Virus could only be demonstrated in them by inoculating tissue extracts into another strain of mice which carried no virus and were not immune; these afford a rare instance in animal pathology in which the investigator has available an "indicator host". So far as Traub could determine, the changes in the epidemiology of the disease were due to a decreased pathogenicity of the virus for mouse embryonic tissue, permitting more and more mice infected *in utero* to survive into adult life. He had noted that the earlier in life infection occurred, the greater was the virus content of the nasal secretions, and the greater the infectivity of the mice for contacts and the longer the carrier-state. Conditions thus gradually became more and more perfect for ensuring that all mice became carriers and hence that all young mice were infected before birth.

Choriomeningitis affords an extraordinary example of an indigenous virus infection of an animal; for it has been possible to trace to a large extent the evolution of the state of "perfect parasitism" taking place during the course of a few years. The facts have probably been easier to trace because of the circumstance, already mentioned, that mice do not develop readily demonstrable neutralizing antibodies against the virus in question.

(b) *Herpes simplex*.—Fever blisters in man may notoriously be elicited by a number of stimuli, in some persons by "colds" and various fevers, in others by ultra-violet light, menstruation, or eating cheese. It has been the general view that these stimuli act by lighting up a latent infection with the virus of herpes, but the epidemiology of the disease has been lately put into sharper focus by Burnet and Lush (1939). Dodd, Johnston and Buddingh (1938), found that herpes virus was responsible for many cases of infectious stomatitis in young children and Burnet has confirmed this. He also confirmed some observations by Andrewes and Carmichael (1930) and by Brain (1932) indicating that those people who were liable to recurrent attacks of herpes carried potent antibodies in their sera, while adults not so liable had no antibodies. There was a sharp division into two categories, those who had large amounts of antibody and those who had none; people from the less favoured social strata tended to be in the former group. Burnet interprets this and other contributory evidence as follows:—many children, particularly those of poorer families, suffer from herpetic stomatitis before they are 5 years old. In the course of this infection they develop in their sera neutralizing antibodies which were not present before, and the titre of these antibodies then remains fairly stable throughout life. This persistence of antibody is probably associated with persistence of virus; where in the body this lies hidden is uncertain, though Levaditi, Harvier and Nicolau (1922) found virus in the mouth washings of "normal" persons. Burnet suggests that herpes virus lies latent somewhere in the central nervous system, though he himself failed to find it in what he thought a likely spot, the Gasserian ganglion. At any rate it seems that the habitual "herpetiker", as Doerr calls him, carries virus somewhere about him, and that the antibodies associated with his carrier state are

unavailing to protect him from a local herpes eruption when a suitable provoking stimulus occurs. The effective stimulus is different in different subjects and may have to be a strong one such as does not occur often, for some people who have antibodies are only very rarely afflicted by fever blisters.

Herpetic stomatitis has not been recognized, at least not commonly, in adults; the percentage of herpes-carriers or persons with antibodies does not seem to rise after early childhood is passed: apparently human beings become less susceptible to natural infection with herpes after the age of 5, though experimentally most persons, even adults, *can* be infected by intradermal inoculation of large doses of virus. The greater susceptibility of the very young animal to certain viruses may be important in our attempt to unravel this "indigenous virus" question. It may often be obscured by the protective action of maternal antibodies in the animal's early life, and there may in other instances be an increased resistance of young animals apart from such antibodies. It has already been seen, however, that mice infected with choriomeningitis virus *in utero* are more severely affected than those infected after birth. Young mice also are particularly susceptible to influenza virus and the virus of St. Louis encephalitis. Guinea-pigs and rabbits, which are relatively resistant to influenza virus, may be readily infected *in utero* (Woolpert, 1939). Very young chicks infected with Rous sarcoma virus may be laid low by a rapidly fatal generalized disease without neoplastic characters (Duran-Reynals, 1939). Another example of the same phenomenon is the susceptibility of chick embryos to a number of viruses to which the newly hatched chick is resistant. It need not follow that a sudden decline in susceptibility occurs at birth or hatching: in the case of herpes, liability to infection apparently wanes during the first few years of life. It may be noted that young plants and actively growing bacteria also tend to be more susceptible to infection with viruses (or phages) than older ones in less active growth.

(c) *Virus III of rabbits and mouse-pneumonia viruses.*—Rivers and Tillett (1923) found that after a few serial intratesticular passages of human varicella material through rabbits one obtained an acute orchitis, the agent responsible for which was clearly a virus, now known as Virus III. They were soon led to doubt the relation of this virus to varicella; and Andrewes and Miller (1924) recovered the same virus by similar serial passages through rabbit testes, starting with normal human blood. Rivers and Tillett (1924) found that 10–15% of American rabbits were resistant to the virus and about the same percentage had neutralizing antibodies in their sera. They concluded that Virus III was an indigenous virus of rabbits and that it was brought to light by the technique they used. In 1928 I could find no evidence that the virus was present in domestic rabbits in London (Andrewes, 1928), though it has turned up here since, in 1938.

A similar story has to be told now that passage of viruses by intranasal inoculation of mice has become a popular technique. Dochez *et al.* (1937), Gordon, Freeman and Clampitt (1938), and Horsfall and Hahn (1939), have thus brought to light viruses causing pneumonia in mice. The agents described by these authors are not necessarily the same, but in each instance some material or other has been dropped up the noses of mice, their lungs have been later harvested and emulsions thereof dropped up the noses of other mice. After a few passages, pneumonia has appeared and it has been easy to show that this is caused by a virus transmissible in series. Horsfall and Hahn have been able to recover their virus from certain strains of white (Swiss) mice but not from others; all of the Swiss mice originally came from the same source. The strains of mice from which the virus is not recoverable are much less sensitive to it. One may wonder whether their freedom from it is due to an inherent resistance—yet they are presumably very similar genetically; or whether the resistant mice may not have an infection-immunity such as Traub's choriomeningitis mice all had when, in 1937, a stable virus-host equilibrium seemed to be established.

One naturally wonders how this technique of serial passage through apparently normal animals can act to bring to light a wholly hidden virus. Most probably the technique simply permits one to obtain virus in an abnormally high concentration. Some viruses—certain strains of influenza for example—produce a symptomless infection when given in very small doses and typical symptoms when inoculated in larger quantity. The mouse-pneumonia viruses and Virus III may normally produce a subclinical infection and never build up to a high titre. But rapid passage under favourable conditions could conceivably, by keeping them artificially, as it were, in a permanent logarithmic phase of growth, allow them to attain a far higher concentration than is normally possible, a concentration such that they could produce obvious pathological changes and symptoms of disease. Possibly the change is not merely quantitative: the unusually favourable environment may permit a mutation of the virus, though of this we have no evidence. Study of this laboratory phenomenon of the production of a disease from an inapparent infection by rapid passages must make the epidemiologist think of analogies in his field. Here it often seems that provision of opportunities for rapid passage of an agent from one individual to another in nature may create an epidemic out of nothing.

(iv) *Complicated Latent Infections—Swine Influenza*

Let us now consider a latent infection of, perhaps, a special kind. Shope (1939) has recently brought forward evidence which may bear on our theme; he has suggested that the virus of swine influenza may pass through an intermediate host in lungworms. The embryonated ova of lungworms from a pig are passed in the pig's faeces, and some of them are later ingested by earthworms in which the lungworms in question pass some stages in their life-cycle. At a later date the earthworms are eaten by other pigs and the lungworms find their way through the intestinal walls back to the pig's lungs. Now the lungworms in a pig with swine influenza may take up or be contaminated with the swine influenza virus, and their embryonated ova, hatching out after being taken up by earthworms, may carry virus with them through their life-cycle back to another pig when he, in due course, eats the earthworms. The virus-carrying lungworms will thus reach the susceptible tissues of a pig's lungs. But this event is not enough to make a pig go down forthwith with swine influenza. The virus will lie harmlessly in the worms until some provoking stimulus lights the infection up in some quite obscure fashion, and the pig *does* get influenza. An injection into the pig's muscles of a culture of *Hamophilus influenzae suis* has been the provoking stimulus chiefly used. These are Shope's interpretations of his findings; his results need further study and confirmation before rash conclusions are drawn. It appears possible, however, that in this instance we have a virus lying latent not in the host's own tissues, but in those of an animal parasite, in which they are revealed after a provoking stimulus. This provocation does not act, however, merely by mechanical liberation of virus from the lungworm, for Shope has not yet demonstrated virus by injection into pigs of ground-up "infected" lungworms; it would seem possible that virus is present in the worms in some altered state. Shope's stimulating suggestions are noted at this point in order to point out that latency of a virus may be a complicated affair.

LATENT VIRUSES AND CANCER

Theories about cancer which claim a rôle for virus in the aetiology must postulate that such a virus, or viruses, are very widely distributed in the animal kingdom, that they are normally latent infections, but are lit up by some stimulus such as the application of a carcinogenic hydrocarbon. Those who decline seriously to consider such a view do so partly on the ground that a ubiquitous latent infection with a virus is "absurd". Enough has been said in the first part of this address to show that ubiquitous latent virus-infections come into the realm of known facts; and facts are absurd only to those who do not understand them. There is admittedly very little direct concrete evidence that cancer in general is

caused in the manner suggested. But I can draw your attention to some facts in experimental cancer research which seem to me strongly reminiscent of some things we have just discussed in the field of virus-diseases.

(a) *Fowl paralysis* (neurolymphomatosis).—Let us first glance at fowl paralysis, an obscure disease which is certainly related to neoplasms. As you are aware, the paralysis in this disease is associated with great enlargement of the nerves, due to massive infiltration with round cells. Lymphomatous tumours seem often to arise from the collections of round cells associated with the typical lesions; and a strain has been described by Furth (1934) which on propagation yielded sometimes lymphomatous nerve lesions, sometimes myelomatosis and sometimes endothelioma. Dalling stated recently that there were three theories of the nature of fowl paralysis: that it was an infection, a tumour or a virus-disease. While reluctant to introduce theology and particularly the Athanasian creed into pathology, I would suggest that we have here not three explanations but one explanation, not three theories but one theory; an "infection with a tumour virus" would serve as an explanation which could cover all the facts. Evidence that an infective agent was concerned has hitherto been inconclusive, but Blakemore's (1939) recent studies have marked a definite advance. From a stock of fowls subject to fowl paralysis he obtained by in-breeding a strain free from the disease yet highly susceptible to inoculation with infectious material from diseased birds—in fact a perfectly good indicator-strain. Inoculations of fowl paralysis material into his indicator hosts produced either nothing very much or merely "unthriftiness" associated with lesions in the heart and liver and occasionally true fowl paralysis. But further passage raised the virulence of the infective agent and gave rise to an acute disease, the lesions of which were at first inflammatory, later often lymphomatous. Evidence was obtained suggesting that fowl paralysis is the chronic stage of an acute disease which may naturally be wholly or largely symptomless. There is yet no proof that the agent is a virus, but analogy with similar conditions in fowls, such as leukaemia and sarcoma, makes it very probable. It seems fairly certain that the agent can be transmitted from the mother through the egg; there are also reasons for believing that purchased cockerels carrying a latent infection may have passed the disease on to their progeny (Blakemore, 1934–35). There is also evidence that environmental factors may determine whether or not a latent infection will blossom out into declared disease: chicks from infected stock have been divided into two lots and those taken and reared at one farm have developed symptoms while those kept at another have not.

(b) *Tar-sarcomata in fowls*.—Most normal fowls develop as they grow older neutralizing antibodies active against filtrates of Rous sarcoma virus; the antibodies are, however, usually low in titre compared with those which one finds in birds bearing slow-growing tumours. Their presence suggests, though it does not prove, that many normal fowls may carry or have had contact with a virus serologically related to fowl tumour viruses such as that described by Rous. McIntosh (1933) reported that a number of fowls injected intramuscularly with tar developed sarcomata and that in three instances the resulting tumours could be propagated with filtrates. The filtrable agents had the properties of a virus. Recently McIntosh and Selbie (1939) have obtained two more filtrable tumours in tarred fowls arising at the site of inoculation of the tar. No similar tumours appeared in birds of the same age not treated with tar. The suggestion naturally arises that the tar injected into these fowls has activated an indigenous virus of the Rous-virus family. Other workers, however, have produced tar-sarcomata in fowls and have failed to obtain active filtrates. A possible clue to these diverging results is afforded by some work of my own (Andrewes, 1936) and of Foulds (1937). A tar sarcoma was produced in a fowl by Mellanby and this, though transplantable with cell-grafts, has always resisted efforts at transmission with filtrates. The tumour would, however, grow on inoculation into pheasants; sometimes very large tumours

formed. The inoculated pheasants regularly developed neutralizing antibodies active against filtrates of Rous No. 1 sarcoma; injections of normal chick embryos caused no such antibodies to form. Fowls grafted with this tar sarcoma also developed potent antibodies to the Rous virus (Andrewes, 1939). The suggestion is a strong one that the non-filtrable sarcoma contains a virus serologically related to Rous virus though not directly demonstrable by filtration experiments. It must be mentioned that the filtrable agents of various histologically distinct fowl tumours have been shown to be related serologically (Andrewes, 1931) so that cross-neutralization of the kind suggested would not be surprising. Foulds (1937) obtained similar findings with a non-filtrable dibenzanthracene sarcoma in a fowl; he was able to elicit anti-Rous properties in the sera of rabbits by injections of crude or even filtered extracts of this non-filtrable tumour. It may be, therefore, that tumours induced in fowls by injections of tar or other carcinogens may generally owe their continuing malignant character to the action of a tumour-virus within the cells, such a virus having been liberated from some restraint by the poisoning of the cells by the tar. It may be that this liberation from restraint is only sometimes sufficient, as in McIntosh's experiments, to allow the virus to infect normal cells: possibly the virus which we imagine to be carried by McIntosh's strain of fowls is more easily exalted in virulence than the indigenous fowl tumour viruses present in the fowls studied by other workers.

To suggest that a carcinogenic agent can upset a cell-virus equilibrium in the manner postulated is not to theorize entirely beyond the known facts. For as Ahlström and I showed (1938) tar and other carcinogens can apparently act in just such a manner in rabbits infected with Shope's infectious fibroma virus. Normally, this virus causes a proliferation of fibroblasts leading to the formation of sarcoma-like tumours; but these always regress after a few weeks. In rabbits treated with tar the regression is delayed, often for months, the "tumours" become locally invasive and rabbits may even die with generalized fibromatous lesions. Carcinogens do not have this dramatic effect on the course of vaccinia and other virus diseases which are not associated with great proliferation of cells. It may be recalled in this same connexion that another rabbit-virus, the papilloma virus also described by Shope, produces warts on rabbit's ears and that these may become malignant, but only after many months. After previous preparation of the ears with tar, however, the same virus produces growths many of which may be malignant almost from their first appearance (Rous and Kidd, 1938). How the tar acts we do not yet understand: it may do so through an upset of a cell-virus equilibrium such as would seem to explain the phenomena in McIntosh's fowls and in our tarred fibroma rabbits.

#### *Bittner's Experiments with Breast-carcinoma in Mice*

Some strains of mice used in cancer-research have an incidence of breast cancer in breeding females of 80-90%, while in others the incidence is almost nil. In hybrids between two such strains it has been found that the incidence of breast cancer in the offspring depends not upon Mendelian rules but wholly upon whether the mother came from a high or low cancer family: only the mice born from high cancer mothers developed breast cancer in their later life. Bittner (1939) found that mice from the low cancer strain would develop a high proportion of cancer if they were suckled from birth by high cancer-strain mothers. Conversely, mice from high cancer mothers would have quite a low tumour incidence if they were fostered by mothers of the low cancer strain. It appeared as if something affecting the occurrence of cancer in later life was transferred to the young in the mother's milk. But still odder findings were to come: if young mice of the low cancer stock were suckled by high cancer mothers and consequently developed cancer later despite their hitherto unstained family escutcheon, then, on inbreeding them their children and grandchildren were also very liable to breast-cancer: the blot on the escutcheon was passed on. On the other hand,

young of the high cancer stock which escaped the disease through being fostered by low cancer mothers were able to pass on their freedom from cancer to their descendants. Bittner has considered whether this agent transmitted in the milk may not be a hormone; I can swallow some things, but it is hard to swallow a hormone which, when taken in infancy will condemn your grandchildren to the development of cancer in their years of maturity. On the other hand, if we try to explain the phenomena in terms of viruses, if we cast our minds back to some of the instances of indigenous virus infections, we find that choriomeningitis and mouse pneumonia viruses are present almost universally in certain strains of mice but not in others; that choriomeningitis and herpes viruses can apparently infect the very young animal more readily than the adolescent or adult, though it is true that no example of transmission of the agent in the mother's milk was described; that, again in choriomeningitis, the infection may be made manifest only by the giving of a non-specific stimulus in later life. Such references to known phenomena in the virus field make it not too difficult to believe that Bittner's high cancer mice are infected with an indigenous virus, which normally remains quite latent. But ultimately a series of stimuli, perhaps acting over a long period, reveal its rôle as a causative agent of breast carcinoma. It might be expected that an agent with a habitat in the breast might be transmitted in the milk and that mice not infected in their young, highly susceptible state might escape infection when they had grown refractory with increasing age. There is, we may note in passing, evidence that infection through the milk does not occur with other mouse-tumours, notably lung-carcinoma.

*"Toothless Viruses"*

We have in our discussion come across examples of viruses which cannot be demonstrated directly by injection into fresh hosts, but only by roundabout means. The evidence for presence of a virus in non-filtrable sarcomata in fowls was based on serological evidence. Purely immunological, also, is the evidence that a virus is present in most rabbit papillomata in domestic rabbits and in the cancers developing therefrom. While papillomata in wild cottontail rabbits usually yield extracts which will readily infect either wild or tame rabbits, the warts which appear on domestic rabbits do not as a rule do so. Even in extensive, progressively growing, papillomata in domestic rabbits one usually cannot reveal directly by transmission experiments that virus is present: but extracts of the warts will immunize other rabbits effectively when injected intraperitoneally, leaving little doubt that plenty of virus is present in some masked form (Shope, 1937). Again, the carcinomata derived from such warts may be transplanted to other rabbits; when the grafts grow they lead to the development of neutralizing antibodies to papilloma virus in the rabbits' sera—further evidence that an occult virus is present (Kidd, Beard and Rous, 1936). Shope's swine-influenza experiments, also, suggest that virus in the lung-worms is not necessarily present in the normal, fully infective state.

Such findings make one wonder whether a virus may not depend for its power to infect normal cells on some, possibly haptene-like, aggressive mechanism, teeth as it were permitting an entry into the new cell; and whether in certain circumstances a virus may not lose its teeth by disuse-atrophy, as armadillos and ant-eaters have done. This could happen most readily, one may imagine, in the environment of the cancer-cell, where virus could be carried on from cell to daughter and granddaughter cell as cell-division was stimulated and the need to come out of the cell to look for fresh prey would disappear. Viruses which had become toothless by some such process could thus act as a proximate cause for cancer and yet one would never be able to demonstrate their presence by injecting tumour-extracts into fresh hosts. Roundabout methods of revealing them would always be necessary. In the instance of Bittner's mouse-carcinoma the toothlessness would presumably not be absolute; a single incisor would perhaps remain, adequate only to permit entry to the peculiarly susceptible cells of the tender infant mice. It is not too easy to reconcile

the conception of a toothless virus in a cancer-cell with the need for visualizing some possible means for carrying the virus over from one generation to the next. Complete toothlessness would seem to involve a transmission through the germ-plasm: relative toothlessness would allow a little more latitude. Toothless viruses need not be found only in cancers. There are the viruses which, as some workers think, *must* persist after infections and be responsible for keeping up a life-long immunity and yet which obstinately refuse to be demonstrable; may they too not be modified, relatively edentulous, instead of being merely masked by antibody?

Green (1938) has suggested that a cancer-virus in a cancer-cell is the highest conceivable form of parasitism, virus and cell having their division exactly synchronized, almost a virus-host hybridization. I would disagree. The perfect parasitism is rather the association of a latent indigenous virus and its host, neither doing any harm to the other. Such a compromise has probably evolved in more instances than we suspect during the struggles between viruses and their hosts. Cancer, when it occurs in animals, would then be due to the incursion of some unexpected factor, a *tertium quid*, which broke up the happy association; a disease affecting both partners, of advantage to neither. Instead of a disease of man caused by a virus, we should then have to consider human cancer a disease of the man-virus partnership.

I have not attempted to-day to report anything wholly new. I have tried rather to bring together some facts which have impressed my roving eye as it has tried to keep up with a small part of current literature. There are facts about bacteriophages, about plant-viruses, about neurotropic animal viruses, about cancer, which do seem to hang together, to fit into the same sort of general pattern. We have noted that the association of hosts and parasites, particularly viruses, may lead not to violent disease but to a certain balance of power. We have observed that such a balance may at times be upset, with the result that disease occurs after all. Finally we have been led to wonder whether such an upset may not be the fundamental cause of a particular disease, cancer.

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*Discussion.*—Dr. THOMAS LUMSDEN wished to refer to only one statement. Dr. Andrewes had said that Bittner's experiments provided an instance of the transmission of a latent virus since, when young mice of a low cancer strain were nursed by a high cancer-strain mother, the young mice became prone to cancer just as the young of high cancer-strain mice are when normally nursed by their own mother. Dr. Lumsden gathered from talking with Bittner recently that there was no constancy in this side of his experiments. It was generally admitted that the converse was true—namely that young of a high cancer strain, fostered within the first twenty-four to forty-eight hours on low cancer-strain mothers, developed some resistance to the cancer they would naturally have been prone to, but the opposite did not hold and so there was in this case no direct evidence of the transmission of a virus.

Dr. G. M. FINDLAY said that although in one species of animal a virus might produce an active disease, in other species it might be entirely latent. Thus in West and Central Africa about 20-25% of wild monkeys were found to contain immune bodies to yellow fever in their blood. If, however, yellow fever virus was injected into non-immune African monkeys no clinical reaction of any sort occurred. Considerable variation might also occur in the reaction of individuals of the same species to a virus infection. Thus in the majority of persons, Rift Valley fever virus induced a short but unpleasant febrile attack. The speaker, however, had become immune to Rift Valley fever some nine years ago without any sort of clinical attack. An example of a latent virus being excreted in the milk after an acute attack was equine infectious anæmia. Intestinal excretion could also be continued for some time since an instance was known in America where psittacosis virus had been excreted by a parrot for at least eighteen months.

Dr. TOM HARE said that the President had given prominence to certain authors who claimed that fowl paralysis was an infection due solely to a filtrable virus. He, himself, had formed the impression that fowl paralysis, the pathology of which resembled that of neurofibromatosis in man, was inherited as a recessive. Was the President satisfied that in fowl paralysis a virus was transmitted through the germ plasm without explaining how a non-contagious virus was transmitted in mendelian ratios? He (Dr. Hare) contended that the evidence for the inheritance of fowl paralysis could not be ignored. If it should be shown that inheritance did not play the whole part in causing fowl paralysis, he would suggest that if a virus contributed to the neoplastic process it did so only in those birds which inherited a tissue susceptibility.

The PRESIDENT (in reply to Dr. Lumsden) said that his description of the mammary-cancer work had been based on Bittner's published data. Answering Dr. Hare, he declined to elaborate a theory of the inheritance of viruses as recessives until confronted with evidence that such a mode of transmission did in fact exist.

## Section of Otology

President—T. RITCHIE RODGER, M.D.

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[November 3, 1939]

### Syphilis as seen by the Aural Surgeon

#### PRESIDENT'S ADDRESS

By T. RITCHIE RODGER, M.D.

FOR the short address which the President is expected to give on this occasion, I have chosen a subject which for some reason or other has figured very seldom in our discussions during the twenty years of my membership of the Section—Syphilis as seen by the Aural Surgeon.

It may be admitted that this is a subject of diminishing importance because of its diminishing incidence, but cases of syphilis are still of such considerable frequency, and are so easily missed, that it is necessary for us to be reminded from time to time that this underlying constitutional disease may be the explanation of a case which is otherwise difficult to explain.

There are two reasons why the ear and throat surgeon should have the possibility of syphilis continually before him. First of all, his missing of this basal factor in the production of the symptom or symptoms for which he is consulted, would of course render his treatment of these symptoms imperfect and probably fruitless. Secondly, there is a larger and in some respects a more important point of view. These symptoms are but the efflorescence of an underlying constitutional infection, in a sense a fortunate efflorescence, as in a very large proportion of cases, the more usual primary and secondary signs have escaped notice. If we on our part miss these signs, occurring in a region for which we assume responsibility, then the constitutional disease remains untreated; and however important and distressing may be the symptoms for which our aid is sought, they are probably nothing as compared with the possibilities of the more remote future, e.g. general paralysis or tabes.

Nothing has impressed me more in connexion with this subject than the large proportion of cases among women who have had no suspicion of the cause of their symptoms—genuine cases of syphilis insontium (syphilis of the innocent). Probably because they were innocent victims of erring husbands they were not on the look-out for primary and secondary signs as a more sophisticated member of their sex might have been, and they allowed these early signs to pass without seeking advice.

This large number of unsuspected cases leads one to take certain precautions as to making known the diagnosis. It is obvious that on first seeing the patient one has no right to give a diagnosis of syphilis nor even to ask such direct questions as may betray to the patient the trend of one's mind. A declaration of the diagnosis is likely to lead to a considerable domestic upheaval, and we have no right to risk this without being absolutely sure of our ground. My custom has been when the symptoms suggest the possibility of syphilis, to take blood for a Wassermann test on the patient's first visit. The words Wassermann or blood test, however, are forbidden in the Clinic, as so many patients nowadays know their significance. It is generally sufficient to explain that we may get some help from a detailed examination

of the blood. As a rule at this first visit a mixture of potassium iodide and mercury is also prescribed for diagnostic purposes. When the patient returns in one or two weeks we have the Wassermann report to hand, and in the case of mucous membrane lesions, even this short administration of iodide and mercury has in most cases produced an improvement, so that we are in a position to give a definite diagnosis. It is not always considered advisable to do so, however. If a patient is a woman past the child-bearing stage, for instance, it may be justifiable to have her treated without explaining further. Of course, to transfer a patient from hospital to the venereal diseases clinic is tantamount to giving a diagnosis, and to get over this difficulty, in the Hull Royal Infirmary the staff made representations to the Board that one of the syphilologists of the city venereal diseases department should be made an honorary member of the staff, so that he could treat in hospital such patients as we might wish to have treated without declaring the diagnosis.

On the other hand there is the question to be faced whether the patient should not be told frankly and urged to arrange for a blood examination of husband and children.

My special interest in the subject occurred in this way: Some fifteen years ago, when our two Sections met on different days, I used to justify my leaving my practice for a single meeting by putting in also a visit to such of my friends in town as happened to have a clinic on Friday afternoons. Sitting thus one day, with one who like myself is now among the seniors of the Section, I remarked, after a patient had left the room, "What about the possibility of syphilis in that case?" My friend's reply was that he saw syphilis so seldom, the idea had not occurred to him. He was sure he did not see a dozen cases of syphilis in a year.

On my way home I pondered over this remark, feeling sure that I saw many more cases than was the experience of my friend, and it occurred to me that practising in a large seaport, it was possible that I might have a better opportunity than usual of doing some statistical research on the subject.

I began straightway to keep records of the syphilitic cases that came my way, and in ten years had collected notes of 500 cases—including of course a larger number of affections of the throat and nose than of aural syphilis, although it is only of the last named I wish to speak in this Section of Otology.

I ceased the investigation at the end of ten years for two reasons. In the first place 500 cases seemed to be a sufficient number from which to form one's conclusions, and secondly by that time my clinics had undergone some change. With their growth less of the work was under my personal supervision and at the Children's Hospital, where I had seen most of the congenital cases, my junior had taken over the outpatient clinic entirely.

For the purpose of comparison in this address I abstracted the cases diagnosed in both clinics during last year, 1938, and found they amounted to less than half of the yearly average of my ten years ending 1934. This fact deserves some reflection. Does it mean that in these recent years we are at last getting the full benefit of the intensive treatment which has been in vogue for a generation or so? I remember that ten years ago when I was midway in my investigation, I asked an alienist whether he found that his admissions for general paralysis of the insane had diminished since the arsenical treatment of syphilis had been introduced. His reply was in the negative, but he reminded me that general paralysis was as a rule a very late manifestation of syphilis, beginning on the average twenty to twenty-five years after infection, and that we had not yet been using the arsenical injections very generally for that length of time. His successor, however, informs me this year that the number of admissions for general paralysis is now very much reduced.

It is probably true, therefore, that we are now seeing a real reduction in the secondary and tertiary signs of syphilis because of timely and successful treatment of the early stages of the disease.

There is also the question of a diminished incidence of the disease itself.

Dr. Barlee, who is in charge of the city venereal disease department in Hull, has very kindly provided me with statistics covering a period of years, and these show a distinct downward trend. New cases in 1934 for instance, i.e. the end of my ten years' period, numbered 246. In 1938 they had fallen to 150. In 1920 and 1921, immediately after the last war, the figures were 684 and 557 respectively. If history repeats itself, so that we find an increased incidence after the present war, it will not have proved inappropriate that we should be discussing this subject to-day.

All the cases included in the series were in my opinion genuine cases of syphilis. Any doubtful cases were excluded. 94% gave a positive Wassermann and all of the remainder showed unequivocal signs such as interstitial keratitis and Hutchinson's teeth in congenital cases, radiate scarring on the posterior wall of the pharynx, or palatal gaps in acquired cases, or a definite history of infection and treatment. I frankly admit that during my investigation I asked for a blood examination in every case in which I thought there might be the slightest possibility of syphilis, but this is no more than should be our custom at all times. One meets with practitioners who seem to think that when you have asked for a report on the blood and it proves to be negative, you have made a mistake in diagnosis. We might as well make the same accusation against a physician who examined the urine only to find it quite free of albumin or sugar, or who used his stethoscope and had to report that the heart sounds were normal. One conclusion I came to quite early, and it was confirmed more and more as time went on, was that if a case does not fit easily into any of the common categories it is well to exclude the possibility of syphilis; or if what has seemed to be an ordinary case of one of the common conditions does not respond to treatment, again it is well to make sure that an underlying syphilis does not explain the failure. I made it a rule in the wards that if an operation case did not do as well as usual, the blood should be examined. A positive Wassermann is obtained often enough at any rate to justify the rule. It will be remembered that A. J. Wright (1930) drew attention to delayed healing after sinus operations, from the same cause.

The subject of aural syphilis is too large to allow of any attempt to overtake it in detail in a short address. I propose therefore to confine myself to a general review of the ear conditions most commonly encountered, with special reference to their relative frequency. I make no attempt to deal in detail with the pathology of the various conditions.

#### HEREDITARY SYPHILIS IN THE EAR

There is no primary sore in hereditary syphilis. The child as a rule has been infected by the blood-stream of the mother and even *in utero* his blood may be as highly charged with the virus as is the blood of an adult in the efflorescent period of the acquired disease.

We have thus in hereditary syphilis to consider only the secondary and tertiary stages of the disease and the two main classes of ear manifestations correspond more or less with these two stages. French otologists speak of the "Precoce" and the "Tardive" types, just as we speak of the early and late. The Precoce or early type includes not only the meningo-neuritis and meningo-neurolabyrinthitis associated with syphilitic basal meningitis, but also otolabyrinthitis, the effects of which are confined mainly to the labyrinth and the middle ear. These two early types admittedly belong to the secondary stage of syphilis. The Tardive or late type is exemplified chiefly by deafness following interstitial keratitis generally about the age of 9 years, and this is mainly a labyrinthitis. Hutchinson at one time was inclined to include these cases also as belonging to the secondary stage of the disease, on account of the eye and ear conditions being as a rule bilateral. It has to be noted, however, that the affections of the eye and ear are only ultimately bilateral. The lesion begins on one side, and weeks or months later the other is affected. There is thus a marked contrast to the skin conditions of the secondary stage of the acquired disease, in which the rash appears with more or less constant symmetry.

The chief practical difference between the early and the late cases of hereditary syphilitic deafness consists in their effect on speech. In the early cases the destruction of the hearing most frequently occurs before the child has learned to speak, and he is destined to be a deaf-mute, whereas in the late cases, speech has as a rule been established, and although the loss of hearing may be extreme, speech is likely to be maintained, although probably in the monotonous voice peculiar to the very deaf.

It may be convenient at this point to refer further to the relationship between hereditary syphilis and deaf-mutism. The Wassermann test as applied to any series of deaf-mutes is not a completely reliable guide. Everyone knows that it is common enough to obtain a negative Wassermann, and even a second negative after provocative medication, in congenital syphilis when Hutchinson's teeth and interstitial keratitis leave no reasonable doubt as to the condition. Urbantschitch (as quoted by Ramadier) records 14% of positive results in a series of deaf-mutes. In the deaf school in Hull, where we have made a point of securing a blood examination for every entrant if the parents agree, the proportion of positive reactions to the Wassermann test was 19%. We may safely conclude that the actual proportion of syphilitic children in the school is at least 25%, the figure arrived at by G. de Parell (as quoted by Ramadier) and separately, by Beck basing their conclusions on an investigation of the family history as well as the serological tests.

I shall now discuss the early cases a little more fully.

First we have a meningo-neuritis affecting the 8th nerve as part of a syphilitic basal meningitis. Mayer has described post mortem an infection arising in the subarachnoid space and infiltrating the nerve fibres of the acoustic, then the perivascular spaces of the columella, the ganglion spirale, and the organ of Corti. Haik has reported analogous findings.

Secondly we have otolabyrinthitis in which the attack is on the middle ear and labyrinth. The outstanding features are sudden onset, without pain or malaise; a little pus on the pillow may be the first indication. Unfortunately, this is also true of tuberculous otitis media and even occurs sometimes in the more common types of suppuration. It is so common in syphilitic cases, however, that it is a good rule to remember the possibility of syphilis when such an onset occurs. Fournier (1886) stated that a large proportion of the cases of bilateral otitis media in very young children have a syphilitic basis. On the other hand, Brockaert and Cheatle deny this. I agree with the latter view. Members of the Section may remember that a few years ago I presented an analysis of my cases of otorrhœa in children under 3 years of age and stated that 40% were bilateral, not necessarily when first seen, but the second ear requiring treatment some days after the first. Very few of these cases proved to be syphilitic.

In these cases of otolabyrinthitis, the spirochaetes with their predilection for periosteum and perichondrium, probably attack first the mucoperiosteum of the inner wall of the middle ear, and proceed to such an infiltration of the thin plate of bone as leads to definite changes on both aspects of it. On the one side we have an otitis media going on perhaps to suppuration and otorrhœa; on the other side we have invasion of the labyrinth with destruction of the delicate nerve endings and consequent labyrinth deafness. Doubtless the process can occur in all kinds of gradation, with preponderance of effect to one side or the other of the party wall. We have thus a class of case among this early group in which there is no otorrhœa and in which the mischief seems to be confined to the inner ear. Baratoux, in a series of post-mortems, found the lesion confined to the labyrinth in four cases, while 12 showed lesions involving both labyrinth and middle ear.

The most distressing reflection in connexion with these cases of the early type is the fact that nearly always our diagnosis is made in retrospect, and after the internal ear is damaged beyond repair. The child is brought to us because he is making no attempt to speak and is suspected of not being able to hear even loud noises. A story of a previous illness suggestive of a mild meningitis, or of an otorrhœa such as I

have described, leads us to have an examination of the blood and the diagnosis is beyond dispute. In my series I classed four cases as being meningitis and 11 as otolabyrinthitis.

#### DEAFNESS OF LATER ONSET IN HEREDITARY SYPHILIS

When a child, the subject of inherited syphilis, passes through the first few years of life without being affected by any of the ear conditions already referred to, he has still to run his greatest risk as far as the loss of hearing is concerned. The labyrinthitis which, associated with interstitial keratitis and notched teeth, completes the well-known Hutchinson triad, constitutes the largest and most distinctive group of ear affections due to hereditary syphilis. In my series it occurred in 44 out of 121 congenital cases, very many of that total not presenting ear conditions at all, at least during the time of observation.

This most common form of deafness in congenital syphilis is an affection of the labyrinth without any obvious involvement of the middle ear. It may be equally innocent of any involvement of the auditory nerve or meninges, and is never associated with coincident affections of other cranial nerves. As Ramadier (1928) says: "it forms a class as distinctive and as well recognized as any other entity in aural practice, better defined now with our improved means of examining both cochlear and vestibular functions, but sufficiently well recognized at a much earlier date."

Hutchinson (1863), who was the first to define it, wrote: "A form of deafness which occurs in these cases and which as far as what little observation I have made on the subject goes, appears to be peculiar to them, is one in which the function fails without any external disease. It is usually symmetrical, not infrequently its stages are rapidly passed through, and a patient who six months ago could hear almost perfectly, becomes, without otorrhoea and without any marked degree of pain—utterly deaf".

In 1886 Fournier describes the condition in expressive language which it is difficult to translate without losing much of its forcefulness.

"... a profound deafness which one meets with in hereditary syphilis in the second infancy or in adolescence. It comes on without the usual concomita of an otitis, without any objective signs to explain it. It begins suddenly and develops with a rapidity surprising and extraordinary—in a short time reaches a considerable intensity and as a rule persists in that state rebellious to all intervention. The child or adolescent begins to lose the hearing in one ear. From day to day, quite literally, the hearing becomes worse and worse until in some weeks or a month or two months he hears nothing at all in that ear. For a variable period things remain in this state, then the other ear, unaffected till now takes its turn. The hearing declines on that side and with the same rapidity as on the other, and soon it is quite gone, leaving the patient completely deaf. The connexion between this form of deafness and hereditary syphilis is proved by numerous observations in which one has seen it occur among subjects the issue of syphilitic parents as also among subjects presenting in themselves the stigmata of hereditary syphilis."

These descriptions of seventy-five and fifty years ago are accepted to-day as essentially correct.

Both the interstitial keratitis and the deafness are practically always ultimately bilateral. The age of onset of the deafness is generally between 8 and 15 years but it may occur as early as 4 years, and in two of my cases it first appeared at 45 and 47. The deafness as a rule supervenes two or three years after the keratitis; in only one of my cases did it antedate the eye condition. The late J. S. Fraser (1936) also reported one such case.

This form of deafness probably never occurs in the acquired disease. Ramadier found in the literature only three cases attributed to acquired syphilis, but from the histories questioned the accuracy of the allocation. Similarly, Hutchinson recorded

that he had met with only one case of bilateral keratitis in the acquired disease. The cases I have already referred to in which the deafness began at 45 and 47 might have been suspected of being subjects of the acquired form of syphilis but for the obvious signs of past interstitial keratitis and other stigmata of the congenital disease.

All observers agree with Hutchinson that the incidence is twice as great among females as among males. In my series it was exactly so. Tinnitus is a very frequent symptom. The noises may precede the deafness and may persist long after the hearing is completely lost. Vertigo is almost as frequent as tinnitus and occurs in all degrees of severity. One of my patients had fallen downstairs and another was unable to go out alone. Spontaneous nystagmus was noted in two of my cases.

Coincident with the ear symptoms in this class of case there may be syphilitic lesions of the nose, pharynx, or palate, or more frequently there are signs that such lesions have existed, but they are not at all constant like the keratitis. In my series of 44 cases, 15 showed such lesions, while 40 showed unmistakable signs of the eye affection.

#### DIAGNOSIS

The otoscopic appearances are not of much value, and the Eustachian tubes are habitually patent. In Hutchinson's book (1863), already referred to, "Diseases of the Eye and Ear Consequent on Inherited Syphilis", 21 cases are quoted as the basis of his earliest observation of the occurrence of this late form of deafness in hereditary syphilis. In the first 15 he says he had too readily accepted statements of past ear discharge, this being in accord with his expectations. He admits that no examination of the ears was attempted. In the six last cases, however, feeling himself on the track of something not hitherto thought of, he solicited help in a detailed examination of the ear and all these were without any evidence of past or present middle-ear disease. He and his colleague had not our present advantages in the matter of modern tests for acuity of hearing or for recognizing defects of the vestibular apparatus, but he was able to arrive at the conclusion in advance of all others, that "the deafness in these cases is due either to disease of the nerves or of their distribution in the labyrinth".

The functional tests as a rule, however, are more constant. The deafness is of more extreme degree than one expects in children the subjects of a catarrhal or suppurative condition. Ramadier in 39 cases, that is 78 ears, found that 19% of ears could not hear a shout. In my series of 44 cases (88 ears) 29% failed in the same test. The degree of deafness depends largely on how early the case is seen. As Ramadier puts it, "The loss of hearing is always severe or destined to become so".

Bone conduction may be well conserved for low tuning-forks—that is below 128 D.V.—while for the high notes it is not so.

*Vestibule.*—It is almost invariably the rule that the ravages of syphilis in this form of the disease extend to the vestibule as well as to the cochlea. Although the subjective signs of giddiness frequently fail us, either because they have been unnoticed or forgotten by the young patient, it is very seldom that the objective tests fail to indicate a partial or complete abrogation of vestibular function: and if, in a case which otherwise appears to belong to this group, the vestibular tests give a normal response, it is well to re-examine after an interval.

I might quote one case in which the cold caloric test gave absolutely normal reactions on both sides although giddiness was present when the deafness and tinnitus began three months earlier. It was a case of late onset—a woman of 32, with steatiness of both corneæ and a history of blindness at about the age of 16 when she attended the Eye Department for two periods of ten months and six months. The teeth were irregular but not conclusive, and the Wassermann reaction was negative as regards both blood and cerebrospinal fluid, so that the complete retention of the

vestibular function made me place her only with a query mark among the group. I examined her again after an interval of twelve months. The hearing was still further diminished and the cold caloric reaction was totally absent in the right ear, while on the left side it was very much delayed and diminished.

#### EAR CONDITIONS IN ACQUIRED SYPHILIS

The ear conditions met with in acquired syphilis must be dealt with very briefly. I have not seen a primary sore. It is said to have occurred occasionally on the auricle as a result of biting, kissing, or piercing the lobe with an infected instrument. It has also been reported as developing at the orifice of the Eustachian tube from the use of an unclean catheter.

In the secondary stage of the disease, syphilides can occur on the auricle as on any other part of the skin. Moreover, as the epidermis of the external auditory meatus is relatively moist, condylomata may occur there. Three cases in my series presented such condylomata which cleared up quickly under antisymphilitic treatment.

The middle-ear conditions connected with the secondary stage correspond to a large extent with those described for the early congenital cases. There may be seen an ordinary catarrhal or suppurative otitis media dependent on a syphilitic lesion in the throat or nasopharynx. I have notes of seven such cases, and all resolved quickly when the throat lesion responded to the proper treatment.

On the other hand there may be a genuine specific invasion analogous to the otolabyrinthitis already described. The onset is generally sudden and the degree of deafness is out of all proportion to the otoscopic signs. Tinnitus and giddiness are generally severe and bone conduction is as a rule much diminished.

Mastoiditis has been reported by Driesbeque in 1922 (quoted by Ramadier) as occurring in the fifteenth month of the infection and revealing on operation a sequestrum without any pus. The most important ear conditions of the secondary stage, however, in the acquired disease as in the hereditary, are those affecting the perception apparatus. Most authors agree that the nerve affections are twice as numerous as all the other affections combined.

The common condition is a meningo-neurolabyrinthitis, similar to that lesion in the early hereditary form which so often results in deaf-mutism.

As this nerve deafness of the secondary stage is apt to coincide with the period of active treatment, it has sometimes been attributed to arsenical poisoning, so that it has quite commonly been stated that in employing the arsenical preparations one should be on the lookout for the onset of deafness. Ramadier (1928) discusses the question at some length and comes to the conclusion that there may have been some appearance of truth in the argument in the early days of the arsenical treatment before dosage became standardized. In his view, the danger lies in small dosage, which merely suffices to curb the development of the secondary signs and suppress the rash. As the full efflorescence of the secondary signs indicates and coincides with the maximum output of the antibodies which Nature prepares as its own contribution to the fight against the spirochaetes, it is possible that insufficient medication really deprives the tissues of more natural protection than is being artificially supplied. The loss is apt to affect in greater degree the less vascular nerve tissues and thus the auditory nerve suffers, not from arsenical poisoning but from the want of protection against the virus. Ramadier concludes that the drug should not be stopped but rather that it should be pushed.

In the tertiary stage, gummata may occur on the auricle or in the meatus. In two of my cases ulceration in the meatus with the formation of granulations was not unlike the condylomata already referred to as occurring in three cases, but in one of them there was an obvious tertiary lesion on the forehead (*corona veneris*) and in the other, two large healed gaps in the nasal septum. In both cases the ear condition resolved in two weeks under antisymphilitic treatment; in the former the forehead

had also healed although it had previously resisted various local applications over a period of eighteen months. In the middle ear the same conditions may occasionally be found as in the secondary stage—an incidental catarrhal or suppurative condition arising from specific throat affection or a syphilitic otolabyrinthitis, producing both middle-ear and internal-ear signs.

In my series I classed seven cases as otolabyrinthitis, of which four were bilateral and three unilateral.

The mastoid process was affected in two cases. In one the outstanding feature was a thickened adherent periosteum and in the other a sequestrum was present.

By far the most common condition found, however, in the tertiary stage as in the secondary, is a neurolabyrinthitis without any obvious involvement of the middle ear. It may be due to miliary gummata in the endosteum of the labyrinth analogous to that described by Fraser and Mayer in the late hereditary cases, but we await post-mortem confirmation of this.

This neurolabyrinthitis of acquired syphilis is most frequently bilateral. Of 52 cases in my series 39 were bilateral and 13 unilateral. As those 13 remained unilateral all the time they were under observation, it appears that the condition is not uniformly bilateral as in the late hereditary form.

Noises and giddiness were a marked feature in about one-half of the cases. Spontaneous nystagmus was noted three times. The cold caloric test yielded a normal reaction more frequently than not, in marked contrast to the results of this test in the late congenital group. Coincident paralysis, which never occurs in the congenital cases, was noted seven times, three being facial paralysis, three vocal cord, and one ptosis.

In eight cases the neurolabyrinthitis was associated with tabes dorsalis. In one other tabetic the condition may have been an otolabyrinthitis, as on her second visit five weeks after she was first seen an aural discharge had supervened. This patient was so completely deaf that questions had to be communicated to her by writing. She came into the consulting room with a wide unsteady gait and was found to have Argyll Robertson pupils and no knee-jerks. Treatment gave her a dry ear but no restoration of hearing.

To sum up one might say that the key to the understanding of syphilis of the ear is to be found in the well-known predilection of the spirochæte for nerve tissue and periosteum. In almost all of the syphilitic cases that come to us with ear symptoms, the site of the attack is one or other of these structures. The outlook as far as hearing is concerned is the poorest possible, because although energetic treatment may lead to resolution of the gross lesion, during the period of its activity before our intervention takes place, there has already occurred such an inflammatory invasion of the labyrinth as has crushed the delicate nerve endings beyond any hope of repair.

A syphilitic bone lesion in the ear as elsewhere, may resolve under energetic treatment; a neuritis or meningitis may also respond; but when the restricted spaces of the cochlea and vestibule have been invaded by inflammatory products under pressure, there seems to be no hope of recovery of function.

Apart from the cases of coincidental catarrhal or suppurative deafness already referred to, I have seldom seen any improvement at all in the hearing even after prolonged medication.

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*Discussion.*—Mr. WATKYN-THOMAS mentioned a case of a man of 40 who had noticed slight deafness for many years. Suddenly he went nearly completely deaf. No hearing in one ear and raised voice only in the other. There was no tinnitus or vertigo. Bone conduction seemed completely lost. There was no history of syphilis, but there had been some eye trouble in his early teens and all his teeth had been extracted. Fundus examination showed tessellated choroiditis and the Wassermann reaction was positive; there was some improvement on antisyphilitic treatment. This was undoubtedly a late manifestation in a congenital case.

In continental literature frequent reference was made to the dissociation of labyrinth signs in these cases. He had personally seen Hennebert's sign—the "fistula reaction" without a fistula—but to his own satisfaction he had never been able to demonstrate dissociation between the caloric and rotatory tests.

Dr. DAVID NABARRO said that he had been induced to come to that meeting, although not a member of the Section, by the title of the President's Address. He had been interested in congenital syphilis for a number of years, and he was rather intrigued to hear what the President had to say about the ear in hereditary syphilis. He had been agreeably surprised at the wealth of information which Mr. Rodger had accumulated on that interesting aspect of the disease. The Hennebert syndrome, so far as he knew, had received very little attention in this country, and in fact the only paper he could recall on the subject at the moment was that by Mr. Nehemiah Asherson some years ago, written about some of his own (Dr. Nabarro's) cases which he had had the opportunity of studying (*J. Laryng. and Otol.*, 1931, 46, 326).

Several points of interest emerged from the President's remarks, and with most of them his own experience was in accord. But he was happy to say that from that same experience he did not take the pessimistic view which the President had done about the number of cases of absolute deafness which were seen as a result of hereditary syphilis. Perhaps this was explained by the fact that the cases which he himself had seen at the Great Ormond Street Hospital were all young cases, who by the time they reached the age of 20 had been through a course of treatment and were presumably cured so that deafness was not likely to supervene.

During the course of treatment a mother might say of her child that it was becoming deaf. This might be due to other causes than nerve deafness. At his clinic such cases were sent to the ear department, and not infrequently blocks of wax were discovered, and when these were removed the deafness disappeared. In another group of cases the deafness was of catarrhal origin—a middle-ear type—and here again when the child was sent to the ear department and treated from that point of view, the deafness after a time disappeared entirely or became less marked. It was true that there appeared to be certain cases which had a mixed origin, being partly syphilitic and partly catarrhal. These tended to show alternations of deafness and improvement in the hearing.

But the really syphilitic cases had been in his experience very few indeed. Some of them were in elderly patients who had had interstitial keratitis years ago and became deaf at 40 or 45 through not having been properly treated. His contention was that in these days when congenital syphilis was as a rule properly treated, this serious deafness need not be anticipated. The great aim was prevention. If the children were adequately treated over one or two or possibly three years then he believed a very good prognosis could be given as regards the probability of deafness supervening later on.

When a mother was accidentally discovered to be syphilitic, their professional duty was to the children. They must be examined and their blood must be tested. They were at the beginning of their lives, and it was known that if properly treated in their early days these congenital syphilitics could be made useful members of society and free from any disease, and it was their duty as medical men to see that the children were so treated. As to trouble in the family resulting from disclosure, he usually overcame that by saying that the condition of syphilis might well have been passed on from parent to child and did not necessarily involve individual misdoing. Indeed, there was such a thing as third-generation syphilis, as he had proved in an address given some six years ago. He was convinced that the untreated congenital syphilitic mother could have congenital syphilitic children, and therefore he was able to tell the family that this circumstance did not prove anything against the husband, and that the syphilis might have been inherited from parents or even possibly from grandparents. If only it were possible to go further back and have a Wassermann test of every pregnant woman they would get down to bedrock. It would then be possible not only to prevent the untoward results of the mother's syphilis but to ensure that the next generation was free from the disease.

Mr. T. B. LAYTON said that he was very glad that the President had raised the ethical problem in connexion with this subject; it was a point of which they all needed to be reminded from time to time. In the case of the woman of 45 who had some young children he was entirely in agreement with Dr. Nabarro that one should face the possibility of unpleasantness in the family and insist upon blood examinations of the children being made. If, of course, the woman had no children and was not likely to have any, it might be justifiable to refrain from suggesting syphilis as the cause of deafness.

He had also been interested in hearing the President's remarks on the relics of the old method of diagnosis by means of potassium iodide, probably before the days of the Wassermann test. On another point of diagnosis supposing anything went wrong with the laboratory investigation—and it was desired to take another sample of blood—once potassium iodide and mercury had been given one would be faced with the problem of not knowing whether the result was valid or whether to withhold treatment for a month or so.

From the clinical point of view he wanted to speak particularly about one type of case, namely, that of the patient with congenital deafness associated with the eye condition in the young adult. His experience did not fit in with what Mr. Rodger had said, or rather it fitted in with only one group of cases to which he had alluded, and he felt that the explanation was to be found in Dr. Nabarro's remarks. He did not remember the late form of labyrinthitis starting at the age of 10 to 14, but rather from 17 to 20 or 21. The only symptoms were deafness and keratitis. He did not remember ever seeing such a case with Hutchinson's teeth or with any other tertiary syphilitic lesion in the upper respiratory tract. It was possible that there were two groups of cases of congenital syphilis, one comprising such cases as he had just mentioned, and the other such cases as Dr. Nabarro had dealt with coming on at an earlier age and in which the hearing could be saved. The young persons to whom he referred presented very tragic cases; they were particularly fine specimens of humanity becoming affected both with deafness and blindness; and in his experience no treatment had any effect upon the former.

In two cases he had been able to trace the condition back and in both of them the patient was the child of a mother who probably had had an extremely attenuated form of syphilis derived from the husband who had been treated for the condition some years previously and had every reason to suppose at the time he married that he was absolutely cured. One of these had the following history: A man had had syphilis while serving in the Army at some time in the late eighties. He had every reason to believe that he had been cured for some eight years when he married in the early years of the present century. His wife had never shown any symptoms to suggest that she had contracted syphilis, but deafness developed in their child at the age of about 20 just after the last war. On tracing out the case, the mother had a positive Wassermann, as also had the father. In those days injection treatment had not been started, and the father before he married was quite justified in supposing that he was all right and could marry without danger. In such a case the congenital syphilitic would not be saved from eventual deafness by such treatment as Dr. Nabarro had instituted, because the child in early life, up to 17 or 18 years of age, would never have shown any symptoms of deafness or other syphilitic lesion.

The President had spoken of cases which came on at the ages of 32, 45, and 47. He had always thought that a patient was fairly safe once he had got well into the twenties—say 22 or 23—and he wondered whether the patient who developed the condition at the age of 32 really belonged to the same type; and he queried whether cases which were said to have arisen at the ages of 45 and 47 were not those of some other type of deafness. In one such case a positive Wassermann was not obtainable either from the blood or the cerebrospinal fluid. Therefore although the President had rather shocked him from the point of view of the prognosis which he had had occasion to give once or twice, he was not entirely convinced as to these late occurrences, and he desired to know what was the latest age at which these manifestations might be expected.

Mr. E. D. D. DAVIS agreed with Dr. Nabarro that syphilis of the ear was not at all common. Occasionally one saw a congenital syphilitic patient with chronic suppurative of the ear, and in that case the deafness was very marked. It was just like tuberculosis. The cases did not respond to treatment quickly. A similar thing happened in an operation case which did not heal well, and on investigation a positive Wassermann was found.

With regard to cases of nerve deafness he had the impression that in tabes and general paralysis of the insane, nerve deafness was nothing like so common as optic atrophy. At one time he was

interested in nerve deafness in cases of tabes and other types of syphilis, but the cases of nerve deafness proved to be extremely few. It might be that the deafness was obscured by other nervous symptoms which were more important.

Rutin of Vienna stated that it was necessary to be careful in giving the first dose of salvarsan in an early case. He believed that the vomiting and vertigo were due to the toxic effect on the vestibular nerve occurring as a result of a full dose of salvarsan, and he advocated first treating the patient with mercury and potassium iodide and then going on to salvarsan. By so doing he claimed that he was able to eliminate the toxic effect on the vestibular nerve. It is possible that some cases which had been diagnosed as otosclerosis might have had a syphilitic taint. He himself once had a young woman patient, aged about 30, who had typical otosclerosis. Afterwards she went to Lausanne to see an aurist, and to the speaker's surprise the aurist found that she had a positive Wassermann. After that experience he began to investigate his cases of otosclerosis but he never found indications of syphilis in any others. The girl in question had no other signs or history of syphilis whatsoever.

The President had mentioned an infection of the Eustachian tube by the catheter. For his part he doubted whether that had ever occurred, but he had seen cases of syphilitic ulceration of the nasopharynx with a gumma of the palate. Cases of chronic otorrhoea with a marked degree of deafness should be investigated for tuberculosis and for syphilis.

Mr. FLETT said that he had one case in which the diagnosis between arsenical intoxication of the labyrinth and syphilitic infection was in doubt. The patient was the most acute case of secondary syphilis he had seen. He came up with very large tonsils and a temperature of about 103° F. His throat affection went on almost to bilateral quinsy, both drums became very much indrawn, and the patient was exceedingly deaf. After arsenical treatment the deafness became much more pronounced. In view of the local condition of the throat he did not like to catheterize the Eustachian tube, and it was thought that the deafness was probably due to the arsenical treatment. The treatment was stopped temporarily, but the man now was totally deaf. This is in accordance with Ramadier's statement that the antisyphilitic treatment must be continued.

Mr. R. G. MACBETH asked how vertiginous symptoms in syphilitic cases responded to treatment. He was prompted to ask this question from his recollection of a case of a man of 40 who had been treated for congenital syphilis since the age of 12, though deafness had not been pronounced until the age of 20. This man had become progressively and profoundly deaf though not completely so, and a few years ago showed a condition clinically indistinguishable from Ménière's symptom complex. Medical remedies had failed to alleviate the vertigo and he (Mr. Macbeth) had been driven to expose the semicircular canal and inject alcohol. This had proved fairly successful. He wondered how often such drastic measures were found necessary in these cases.

Mr. SCOTT STEVENSON said that he had suspected for a long time that many cases of nerve deafness, even though the incidence of syphilis was diminishing, were really of syphilitic origin. He was inclined to agree that some cases that appeared in the thirties and forties were of congenital origin. He only wished that it was possible to carry out here the measure which Jackson carried out in Philadelphia, namely, to have the Wassermann test done as a routine in the case of every patient admitted to the clinic.

The PRESIDENT said that he had seen the dissociation of labyrinth signs of which Mr. Watkyn-Thomas had spoken. Indeed the one constant thing about the labyrinth signs of syphilis was their inconstancy. In the other conditions affecting the labyrinth it was usual to find that the functional signs corresponded to the objective signs and the objective signs as regards rotation would correspond more or less with those of the other tests. But in syphilis this dissociation of signs was frequently observed. With regard to Hennebert's "fistula" sign he had tried it for some time, but he was never satisfied that he got a good-going nystagmus. Ramadier and others took quite a different view, however.

Some otologists were so impressed with the loss of bone conduction as one of the earliest possible signs that they propounded the idea that the otologist could often diagnose syphilis in a patient earlier than any other branch of the profession merely by making the bone-conduction test. That, however, was rather far-fetched.

He was very pleased to hear that Dr. Nabarro was not so pessimistic on this subject as he was himself, but his own view was supported by most of the continental writers so far as he had been

able to read their papers. There might be cases of mixed deafness, partly catarrhal, of the kind which he had carefully referred to two or three times in his address, where the deafness was really of a passing type, due to a mucous membrane syphilitic lesion in the nasopharynx.

He fully agreed with Dr. Nabarro in what he had said about their duty to the children, but he was not sure that he agreed with his statement concerning third-generation syphilis. He had not had the opportunities Dr. Nabarro had had of studying the subject, but he had been interested in it and examined it as far as opportunity offered. Hutchinson, of course, did not believe in third-generation syphilis. He thought that was the opinion he finally held after coming across several cases in which he suspected he was on the track of it. He himself had been on the lookout for it, and he remembered one case in which a mother came with a child with the early type of congenital syphilis. The child showed deafness, and the mother herself had interstitial keratitis with a positive Wassermann. He did his best to get the husband along in order to have him examined, because, if he had been negative, there would have been a strong presumption of third-generation syphilis in the case. But the fact that the husband was so shy about coming in spite of his wife's endeavours to get him to come rather suggested that he might be the subject of syphilis himself and that the child had inherited from that side.

He had been interested to learn that Mr. Layton did not find the ages of onset the same as those which he had quoted. It was generally accepted in the literature that the late form of ear affection began most frequently between the ages of 8 and 15 years, though there were examples of the condition at later ages, of which he had himself quoted two or three. With regard to the cases at an advanced age, 45 and 47, which he had quoted, these had distinctive signs of the congenital condition. That raised a question which Dr. Nabarro might be able to answer better than himself, namely, as to whether a congenital syphilitic man or woman might also contract the acquired disease. There was a difference of opinion about that.

Dr. NABARRO (in reply to the President) said that he had seen a case of a woman aged 38 who developed congenital general paralysis of the insane; so that presumably ear trouble might come on at the same age, or possibly even later. He was quite certain that this woman, who had never been treated before, was herself a congenital case, and she had a child with the congenital disease. She had also a brother who had interstitial keratitis at the age of 36.

The PRESIDENT, proceeding with his reply, said that Mr. Davis had raised the question of tabes; owing to shortness of time he had refrained from referring to tabes in his paper. He had nine cases of deafness associated with tabes, and eight of them were the ordinary neurolabyrinthitis of the acquired condition while the other corresponded to the otolabyrinthitis type. This last case, that of a woman, came to him completely deaf, so that all communications with her had to be in writing. She came into his consulting room with a wide staggering gait, she had Argyll Robertson pupils, and loss of knee-jerks—a typical tabetic. He looked upon her as a case of nerve deafness. When she paid her second visit the ears were discharging profusely. He put the case down as one of otolabyrinthitis, but this was not a common condition.

Mr. Flett had mentioned arsenical poisoning. He had not had any cases in which arsenical poisoning was suspected; what he had stated on this subject was quoted from a very well-reasoned monograph by Ramadier entitled *La Syphilis de l'Oreille*<sup>1</sup>, in which he had gone into the question in a manner that had satisfied him at any rate as being very sound.

Mr. Macbeth had asked about vertigo. He had had all degrees of giddiness, including cases of people who could not walk without assistance or who fell downstairs. The giddiness passed off just as it did in Ménière's disease and in the other cases in which vertigo was a symptom. He thought it was equally amenable to improvement by the ordinary drugs that one used. The giddiness responded to treatment, it was the deafness which one apparently could not cure at all by any measures. The treatments employed were all given with the aim of suppressing excitability, not of restoring function.

Mr. Scott Stevenson had given him his support with regard to the interpretation of the late cases. He agreed with him that it would save a great deal of trouble if a Wassermann could be taken every time a patient came inside the doors, either of our private consulting rooms, or of our clinics. But there again it would not be the last word in the matter. It was generally held that if all congenital syphilitics were taken of all ages not more than two-thirds would be found to give a positive Wassermann.

<sup>1</sup> *Bull. méd. Paris*, 1922, 36, 965-969.

## Section of Therapeutics and Pharmacology

President—Sir WILLIAM WILLCOX, K.C.I.E., C.B., C.M.G., M.D., F.R.C.P.Lond.

[October 10, 1939]

### Pharmacy and Pharmacology

#### PRESIDENT'S ADDRESS

By Sir WILLIAM WILLCOX, K.C.I.E., C.B., C.M.G., M.D.,  
F.R.C.P.Lond.

*(Consulting Physician to St. Mary's Hospital)*

My choice of the subject of my Presidential Address was inspired by the fact that for the past thirty years it has been my privilege to be "The Visitor" for the Privy Council to the Examinations of the Pharmaceutical Society of Great Britain which are held in London. The duties of "The Visitor" consist of twelve visits to the examinations yearly and an annual report on the examinations. In addition, "The Visitor" is often consulted by the Society with reference to proposed changes in the curriculum and examinations. Owing to the happy relations which have always existed between us, it has been my pleasure and privilege to have been intimately associated with the great advances in pharmacy of the past thirty years.

From its very inception pharmacy has been closely linked with medicine and shared its history from the earliest times, the stage of magic and priestcraft, to the present day when both medicine and pharmacy have evolved into independent scientific disciplines. Even in very early times in Egypt pharmacy had attained a relatively high degree of development; records as early as 2000 B.C. give evidence of this. In other countries too pharmacy developed early. Arabia, Persia, China, India, and Japan, all contributed. Greece too was an early contributor. Hippocrates (460 B.C.), the "Father of Medicine", was both a physician and pharmacist. He actually described some 400 pharmaceutical remedies, and made and used fomentations, poultices, pills, lozenges, gargles, suppositories, ointments, collyria and inhalations, &c. A great physician, he realized the value of diet and physical remedies such as fresh air, climate, hydrotherapy, massage, &c. The "Hippocratic Oath", still a model of professional conduct for physicians and pharmacists, shows how high was his standard of medical ethics. Galen (A.D. 130), another Greek, wrote many books on pharmacy and medicine, and placed on record many prescriptions. The term "Galenical", denoting a vegetable remedy, is derived from his name. He introduced such things as aloes, colocynth, hiera, cold cream, &c. He was the first to use special clays as remedies for intestinal diseases and poisons, a special variety being terra sigillata from Lemnos. These clays were identical with kaolin, which is such a valuable remedy at the present time for its absorbent and adsorbent properties.

Coincident with the development of pharmacy toxicology took its rise. The experiments of Mithridates (first century B.C.), King of Pontus, in toxicology, are

well known, as also is the failure of his last toxicological experiment on himself. Poison as a means of capital punishment has at least one famous victim, Socrates (402 B.C.) being compelled to drink hemlock juice, the official Athenian State poison.

The development of pharmacy in England presents many features of interest not only in relation to pharmacy itself, but also to medicine and medical teaching. Up to the fifteenth century pharmacy was closely interwoven with medicine and was practised by medical men, by members of the religious bodies and also by those practising alchemy. However, there appeared to have developed after the Norman Conquest a class of persons who sold drugs, for in 1180 the "Guild of Pepperers" was incorporated and this included drug sellers. About this date the term "Apothecary" came into use to include drug sellers and was synonymous with pharmacist. The Pepperers' Guild was succeeded by the Spicers' Guild and this was succeeded and absorbed by the Grocers' Guild which was incorporated in 1345 though it did not receive its Charter till a century later. In 1345 Edward III granted a pension of sixpence a day for life to Coursus de Gangland, an "Apothecary" (note the term) of London for services rendered during an illness of the king. A few years before this, at the death of Robert the Bruce, a record was made of payment to "John the Apothecary" for supplying embalming materials. The granting of a Charter by Henry VIII to the Royal College of Physicians in 1518 and to the Barber Surgeons Company in 1540 placed Practitioners of Medicine and Surgery under the control of these respective bodies. From this time pharmacy began to be separated from the practice of medicine and surgery.

The relationship between the physician and the pharmacist has been well defined by the following: "The function of the Physician is to diagnose disease and to recommend the appropriate treatment. The Pharmacist prepares the remedies recommended."

The relationship between the physician and the pharmacist was somewhat vulgarly described in 1540 by Dr. Bulleyn, a physician, who practised in the reign of Henry VIII and was said to be a cousin of Anne Boleyn:—

"The apothecary must first serve God; foresee the end, be cleanly, and pity the poor. His place of dwelling and shop must be cleanly, to please the senses withal. His garden must be at hand with plenty of herbs, seeds and roots. He must read Dioscorides. He must have his mortars, stills, pots, filters, glasses, boxes, clean and sweet. He must have two places in his shop, one most clean for phisic and the base place for chirurgic stuff. He is neither to decrease nor diminish the physician's prescriptions. He is neither to buy nor sell rotten drugs. He must be able to open well a vein, for to help pleurisy. He is to meddle only in his own vocation, and to remember that his office is only to be the physician's cook." (Charles H. La Wall, "Four Thousand Years of Pharmacy", Philadelphia, 1927.)

The differentiation between pharmacy and medicine and surgery became more acute with the official recognition of the apothecary. In 1617 the Apothecaries' Guild, which had previously been incorporated with the Grocers' Company, petitioned for a separate Charter which was granted by James I, the body being known henceforth as "The Worshipful Society of Apothecaries of London." The members of the Society of Apothecaries sold drugs and dispensed medicine on the prescriptions of the physicians or surgeons. They were in fact pharmacists. In addition, they appear to have in many cases acted as (unqualified) assistants to medical practitioners, performing minor operations such as blood-letting, bandaging, splinting, and administering medical remedies such as enemata, &c. It is not surprising that very soon some of the members of the Apothecaries' Society began not only to sell drugs and practise as pharmacists, but also to visit sick persons and prescribe medicines; in other words they began to practise as general medical practitioners as well as pharmacists. This led to continued conflicts with the Royal College of Physicians which went on for many years.

James I was always a staunch supporter of the Society of Apothecaries and took the greatest interest in it, no doubt partly due to the fact that Dr. Gideon de Laune, the founder of the Society, was the physician to his queen. In 1622, the Grocers' Company, jealous of the progress of the Society of Apothecaries, petitioned the king that the charter should be taken from the apothecaries and that they should return to the fold of the Grocers' Company. To this appeal the king valiantly replied "Grocers are but merchants, but the business of the apothecary is a mystery, wherefore I think it is fitting they should be a corporation of themselves".

By using the term "mystery" he implied that the business of the apothecary was a science or profession requiring study and research. Similarly the phrase "art and mystery" of the apothecary used in the Charter of the Society means "The Art and Profession of Pharmacy".

The case of Joseph Lane derived from the ancient records of the Society of Apothecaries and transcribed by my friend Dr. Cecil Wall, Archivist of the Society, illustrates some of the activities of the Society in these early days.

#### THE CASE OF JOSEPH LANE

In May 1632, Charles I ordered the Royal College of Physicians to make an inquiry into the cause of death of Joseph Lane. Cromwell, his servant, had been accused of poisoning his master and had been sentenced to death, but constantly denied his guilt.

The College of Physicians conducted an inquiry, from which it was proved that Joseph Lane was suffering from severe constipation. He was attended by Christopher Matthews, an apothecary, who ordered a draught containing rhubarb and senna. This was followed by seven or eight actions of the bowel, but thinking that the purgative was not sufficiently complete, a bolus of calomel was ordered. This was obtained by Cromwell from the shop of an apothecary named Bacon, and was said not to have been properly gilded over.

When the bolus was taken severe vomiting and violent diarrhoea occurred, the stools containing blood. Death followed as a result of these symptoms. A post-mortem examination was conducted by the College, and it was found that death was not due to natural causes, but to corrosive poisoning. The probable cause of death was poisoning by perchloride of mercury (corrosive sublimate) present as an impurity of the calomel contained in the bolus.

In those days there was no adequate supervision of the sale of drugs, and calomel frequently contained an appreciable amount of the poisonous perchloride of mercury (corrosive sublimate) as an impurity.

Cromwell was acquitted as a result of the inquiry. An interesting sidelight on this case was the recommendation in the Report that certain poisons such as arsenic, corrosive sublimate, opium, &c., should only be sold to persons known to the vendor and the reason of the purchase stated—a recommendation which only became law on the passing of the Pharmacy Act of 1868, i.e. over two hundred years later.

During the first century of its existence the Society of Apothecaries had to contend with the jealousy and rivalry of the more ancient College of Physicians (founded in 1518). This rivalry is well illustrated in the recommendation that no apothecary should be allowed to prescribe or supply medicine except on the prescription of a living physician. Since at that time only fifty fully qualified physicians existed in a London with a population of at least a quarter of a million, when disease was much more rampant and much more severe than at the present day with our improvements in sanitation and hygiene, such a recommendation was obviously impracticable of adoption.

In their conflicts with the College of Physicians the Society of Apothecaries always received the full support of the public since they supplied a very pressing need. This

support was enhanced by the action of the members of the Society during the Black Plague of London in 1665 when they manfully stuck to their posts and did their utmost for the sick and dying.

The test case in the year 1703 finally settled the rivalry. In that year a member of the Society of Apothecaries, named Rose, gave advice and treatment to a patient without calling in a physician. The facts were admitted and Rose was convicted. The Society took the case to the House of Lords where the verdict was reversed on the ground "that both custom, and the public need required that Apothecaries should be allowed to advise their clients as well as to treat them". This decision has been termed the "charter of the general practitioner".

The Apothecaries of the Society have since 1700, and probably before, ceased to practise as chemists and druggists and pharmacists except that they may have dispensed the medicines of their own prescriptions.

Throughout its career the Society of Apothecaries has done much to advance the study of botany. The Apothecaries' Garden in Chelsea was founded in 1673 and was afterwards given to the Society by Sir Hans Sloane. By its encouragement of the development of provincial medical schools and by the requirement of evidence of adequate instruction before the candidates sit for their Diploma, the Society has done much to advance medical education, and official recognition of the Society as a diploma-granting body was established by the Apothecaries Act of 1815—L.S.A. In 1858 the General Medical Council was established and accepted the diploma of the Society (L.S.A.) for registration. With the Medical Act, passed in 1886, which required evidence of qualification in medicine, surgery, and midwifery, before admission to the Medical Register, the Society of Apothecaries was given permission to conduct its examination in the three required subjects and to grant a registrable diploma with full privilege. The title of the Society's licence was changed to L.M.S.S.A.

*The Pharmacists* manfully continued to function as pharmacists and chemists and druggists until 1841, when active steps were taken to obtain Government recognition and protection. In that year the Pharmaceutical Society was founded, Mr. William Allen, F.R.S., being its first President, and a Charter was granted in 1843. The first "Pharmacy Act" was passed in 1852. This Act gave powers to the Council of the Pharmaceutical Society as regards registration of pharmacists and to conduct examinations for qualification of persons before admission to the Register. In 1868 a "Pharmacy Act" was passed which regulated the sale of poisons and gave a schedule of poisons. This was followed in 1908 by a more comprehensive "Poisons and Pharmacy Act" with an improved schedule. In 1920 *et seq.* the Dangerous Drugs Acts were passed and these came under the control of the Home Office. In 1933, after a very thorough review by a special departmental committee, of all the laws and regulations relating to "Poisons and Pharmacy", a new and comprehensive Act was passed, previous acts and regulations being annulled. The laws regarding dangerous drugs (of addiction) came under a separate category under the control of the Home Office as already mentioned.

*The Privy Council*, up to 1933, had been the controlling authority of all matters relating to poisons and pharmacy, and had been advised by the Pharmaceutical Society regarding any alterations or additions required in the Schedule of Poisons. It was decided that the Privy Council should remain the controlling authority on all domestic matters concerning the Pharmaceutical Society, such as constitution, registration powers, educational and disciplinary functions. But it was decided that the control of the sale of poisons should be transferred to the Home Office.

*The Poisons Board* was established by the Act of 1933, as the "Advisory Committee" to the Home Secretary regarding poisons. It was to consist of sixteen members with powers to appoint additional members not exceeding three in all.

The Poisons Board was constituted in November 1933 and represents those Government departments and scientific organizations specially concerned with poisons. At the present time it consists of nineteen members :—

- 4 appointed by the Secretary of State for the Home Office.
- 1 appointed by the Secretary of State for Scotland.
- 2 appointed by the Minister of Health.
- 1 appointed by the Minister of Agriculture and Fisheries.
- 5 appointed by the Pharmaceutical Society of Great Britain.
- 1 appointed by the Royal College of Physicians of London.
- 1 appointed by the Royal College of Physicians of Edinburgh.
- 1 appointed by the General Medical Council.
- 1 appointed by the Council of the Institute of Chemistry of Great Britain and Ireland.
- 1 appointed by the British Medical Association.
- 1 *ex officio* representing the Government Chemist.

The Poisons Board is thus seen to be representative of those bodies specially concerned with the control of poisons, and is a guarantee that as far as possible the interests of the public will be safeguarded.

From 1868 (the date of the first Act regulating poisons) up to 1933 the Pharmaceutical Society of Great Britain had been the advisory body to the Privy Council which was the controlling authority regarding poisons; the control of the dangerous drugs of addiction was taken over by the Home Office in 1920.

The Departmental Committee on the Poisons and Pharmacy Acts of which it was my privilege to be a member, made a very searching inquiry and issued its report on January 14, 1930. It was on this report that the comprehensive "Poisons and Pharmacy Act" of 1933 was based. At the inquiry it was unanimously appreciated and agreed that from 1868 to 1933 the Pharmaceutical Society had performed the responsible duties as the advisory body to the Privy Council in an admirable and impartial manner. In view of the great advances in pharmacology, chemistry, pharmacy, and scientific research generally, it was felt that for the future the Home Office would be more fitted by its constitution and powers to become the controlling authority regarding poisons, a responsibility from which the Privy Council welcomed release. It was felt that the control of poisons and all matters relating to them required an advisory body of wide interests, representative not only of Government departments specially concerned, but also of pharmacy, medicine, pharmacology, and chemistry.

The Pharmaceutical Society were whole-heartedly in agreement with the appointment of the Poisons Board as representative of the wide interests concerning poisons, thus replacing them as the advisory body to the controlling Government department—the Home Office. The Poisons Board was constituted in November 1933, and as a result of their keen interest and the arduous work of many lengthy meetings the Report of the Poisons Board was delivered on May 29, 1935. The statutory rules and orders based on this report were published on December 21, 1935, under the authority of the Home Secretary, Sir John Simon. These came into force on May 1, 1936. Under the newly established machinery facilities were provided for the modification and additions to the schedules of poisons and for the issue of statutory rules and orders as required. Unnecessary delay was avoided and the rapid advances in pharmacology, pharmacy, and chemical research were fully provided for.

#### *Modern Advances in Pharmacology*

Up to the beginning of this century the resources of pharmacy were little different from those of the preceding two hundred years. Since this, however, enormous advances have been made and progress has been by geometrical rather than arithmetical progression. The co-operation of the chemist, the pharmacologist, and the physiologist had produced compounds of great therapeutic importance.

By the free use of animals for testing out the therapeutic effect of the newly discovered compounds an enormous number of new drugs has been added to the armamentarium of the pharmacist. Among these are included salvarsan and its modifications, the barbiturates and their host of modifications, sulphanilamide and its many derivatives, insulin, and zinc protamine insulin. Many of the vitamins have now been isolated and their chemical constitution determined. Many of the hormones and active principles have not only been isolated but synthesized, and artificial products with a similar activity have also been produced (oestrogenically active stilboestrols).

By the establishment of the Pharmacological Laboratories for research into biological standardization and the standardization of various medicinal substances which cannot be adequately tested by chemical methods the Pharmaceutical Society has performed a valuable service to pharmacological research.

#### *Educational Aspects in Pharmacy*

The great advances in medicine and therapeutics have naturally increased the amount of training necessary for the modern pharmacist if he is to carry out his new duties adequately, and the Pharmaceutical Society has altered its curricula and examinations accordingly. Up to 1926 the qualifying examination had consisted of oral and practical examinations, but in this year a preliminary examination in chemistry, physics, and botany was constituted as a separate examination. Three years later written and practical examinations in the subjects of the preliminary and final examinations were introduced. Later bacteriology and physiology were introduced into the curriculum for the final examination, while zoology was added to the botany of the preliminary scientific examination. For the qualification of "Pharmaceutical Chemist" a higher examination in the subjects of the final examination is held. In 1937 the Pharmaceutical Society established special diplomas in biochemical analysis and in pharmaceutical analysis. Similarly the University of London has shown its interest in and encouragement of pharmacy by instituting in 1924 the degree of Bachelor of Pharmacy.

#### *Conclusions*

Pharmacy has not lagged behind but has adapted itself to changed requirements and has kept itself in the van of medical progress. The Pharmaceutical Society as the controlling organization has enlarged its educational outlook and by addition to its curricula and the conduct of its examinations has kept in close touch with the similar advances made by medicine through the General Medical Council. Pharmacy is playing its part in co-operating with medicine and pharmacology by assisting in the therapeutic application of remedies which require special training and skill in their preparation.

To conclude, may I again quote the words of James I: "Grocers are but merchants, the business of the Apothecary is a mystery." It is by placing the "mystery" or scientific side of pharmacy completely in the forefront of its aspirations that pharmacy will receive its due recognition as a profession, and be recognized as a branch of medicine whose object is the Public Weal.

## Section of Physical Medicine

President—JAMES MENNELL, M.D.

[November 17, 1939]

### Foot-gear

#### PRESIDENT'S ADDRESS

By JAMES MENNELL, M.D.

WHEN we look at the windows of any ordinary shoe shop of even moderate size we find them labelled "Ladies and Children" on one side and "Gents" on the other. Now why should this be so? "Children" and "Adults" one could understand, but "Ladies" and "Children" together, "Ladies" and "Gents" contrasted and divorced from one another can have no scientific justification. A human foot is a human foot—and one of the most wonderful works of the Almighty at that—be it male or female, nor do the marvels of its design include one pattern for men and another for women. Indeed we need a micrometer screw to distinguish between some of the bones of the skeleton of the male and female foot, while most of them are quite indistinguishable. There can therefore be no particle of scientific justification for the divorce of the two departments that deal with foot-gear. In other words we are the slaves of fashion and of the shoe-manufacturer.

In the windows of one side of the shops we see shoes with relatively high heels and pointed toes, in the other we see them with relatively low heels and square toes. In the section of the first reserved for children we find a reversion to the low heel square toe variety again, but of different type. One can only meditate sadly on the changes that are supposed to take place in the human foot during adolescence to justify the change—possibly in a few months—from the children's type of foot-gear to one of the other types.

*The sole.*—Few people will deny that the heel is narrower than the fore-part of the foot at the level of the heads of the metatarsals, and it is perfectly plain therefore that the general contour of any shoe to be accurate must be roughly triangular when seen from above, the angle at the heel being somewhat rounded (fig. 1). It is not by any means always found that even this elementary law of divergence of these lines is fulfilled adequately and the divergence of the two side lines of the shoe from the centre line of the foot is one that should receive far more attention than is apparently given to it.

The variation in shape and manufacture can easily be tested by placing a straight line between the centre of the heel and the forepart of the shoe running along the middle of the waist. In the tracing of the ordinary human foot this centre line usually passes through about the space between the second and the middle toe, but in many shoes that are made it is quite easy to establish that the design of the last is such that this line passes much too obliquely one way or the other, which means that there must be a very forcible strain upon the joints of the fore-part of the foot tending to swing it round away from the side which encounters pressure. The point where most of this particular pressure is felt is over the head of the first metatarsal on the inner side or the fifth on the outer. In choosing a shoe for a normal foot it is therefore wise to select one that has been built upon a last which gives the appearance, when viewed from the sole, of being thrown well in to the inner side at this point, provided the outer side is not thrown inwards to match (fig. 2).

We now come to the consideration of what should be done with the part of the shoe that concerns the foot in front of the metatarsal heads. We must remember in the first place that the laws of physics apply as much in the fitting of a shoe as in any other branch of life. When we raise the heel from the ground the sole of the shoe must of necessity assume a curve, and if the heel of the shoe remains in contact with the heel of the individual, the fore-part of the foot must of necessity slip forward on the surface of the sole (fig. 3). If it did not do so we should have in action the "three-ply system", and as you know this simply means that three very flexible pieces of wood when bound together become relatively rigid. The allowance that should be made for this sliding forwards is two boot sizes; this may

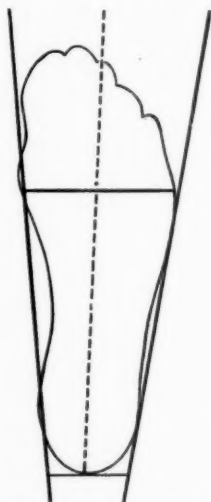


FIG. 1.—To show how the lines joining the sides of the heels to the sides of the metatarsal heads converge towards the heel. Note that although the tracing shows the contour of a foot that is fully serviceable and free from symptoms, it shows nevertheless signs of deformity due to inadequate foot-gear in the cramping of the big toe outwards and of the little toe inwards.



FIG. 2.—Tracings of three welts and the foot for which the shoes are provided. The firm lines show the outline of a plain walking shoe, the broken line that of a (supposedly) similar shoe, and the dotted line that of a golf shoe. The broken line shows the only good fitting. Note the swing away from the centre line of inner side of this shoe and compare it with that of the dotted line. The latter is a far larger shoe but it holds the whole foot in a faulty position.

sound excessive, but in practice it is no more than is essential.<sup>1</sup> Having made this allowance it is quite immaterial what happens beyond it, and the front of the shoe beyond the extreme limit of the essential space is a matter of indifference. Any shape is permissible provided always that the inner side of the shoe does not begin to slope outwards until the front of the big toe is reached, and that any slope which is given on the outer side does not detract from the two boot-sizes which are left for the sliding forward of the little toe on the sole when it bends. There should therefore be no quarrel between the human foot and the pointed shoe, the only thing that matters is where the slopes of the point take place (fig. 4).

*The heel.*—Now we must return to the question of the heel and whether the relatively high heel in the women's department, or the low heel of the men's

<sup>1</sup> A boot-size in this country is  $\frac{1}{2}$  in.

department is the right and proper one. At first sight the temptation is to condemn the one and laud the other, but this simply ignores the one consideration on which alone the solution of this much-discussed problem depends. The point in question is nothing more or less than the elasticity of the calf muscles. If there is any relative insufficiency in this elasticity or in the length of the tendo Achillis, then it becomes obvious that, in order to place toe and heel on the ground together and to keep the leg vertical, something has got to stretch. That "something" is of course the structures in the sole of the foot. Bad as no doubt this may be for the intrinsic structures, the stretching forward of the fore-part must of necessity separate the head of the first metatarsal from the back of the internal malleolus round which passes the tendon of the flexor longus hallucis. If the slide forward of the phalanx of the big toe is great, this muscle or its tendon may lack sufficient

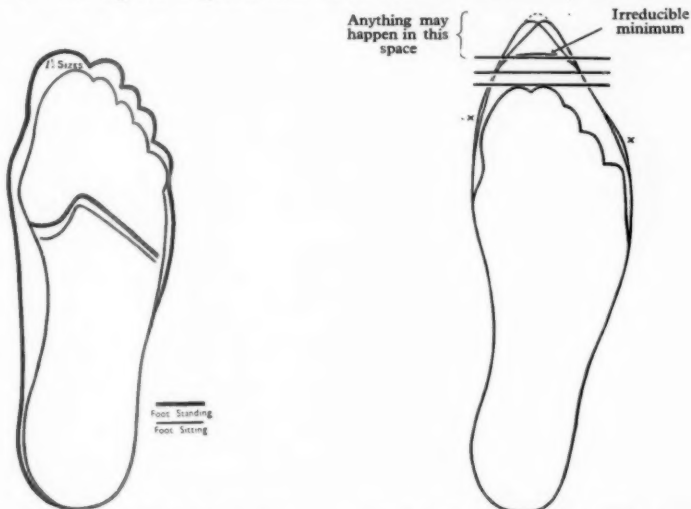


FIG. 3.—Sketch of a foot sitting and standing, showing outline of the back of the metatarsal heads. Note also increase of breadth. This foot is less deformed by foot-gear than that shown in fig. 1 on the outer side, but the big toe still shows signs of pressure. When the heel is raised the front of the toe shoots forwards on the sole to at least the extent of another two sizes— $\frac{2}{3}$  of an inch.

FIG. 4.—To show how a point may be added to an otherwise perfect shoe merely by increase of length. The only disadvantage is the liability to crease rather markedly, and, should the "break" occur at an unfortunate spot—it must occur somewhere—detrimental pressure on the toes may ensue. XX Two points at which the slope commonly begins prematurely. The outer of the two lines shows the correct contour.

elasticity on the one hand or length on the other to allow the distal phalanx to dorsiflex in freedom. Indeed it is almost inevitable that it should be dragged down into the sole of the shoe until, as often happens, a definite hole is dug by the toe on the surface of the sole. Naturally if the sole is thin there is no substance into which the toe can "bed" itself and therefore the tendon becomes more and more strained.

Every normal foot in which the mobility of the joints is not destroyed will elongate to a certain extent when the weight of the body is placed upon it. The extent to which this happens varies considerably, a single boot-size is about the usual, one and a half sizes is not uncommon in a perfectly normal foot (as shown in fig. 3), but anything beyond this should be regarded as pathological.

These considerations lead us therefore to the consideration of the height of the heel required for an individual, be he male or female. The position may be summed

up in few words; there are many men who will never know what it is to walk in comfort with less than two and a half inches of elevation at the heel, while some women (particularly those with a hallux rigidus) can never know comfort with any elevation above about three-quarters of an inch. It is utterly wrong therefore for men to scoff at the relatively high heel of the shoes in the female shop, but equally many women would be well advised to adopt the relatively low heel in the men's shop. I do not wish this to be taken as being in any way a defence for the extravagantly high heel—i.e. anything above three inches—seen in the women's section, for which there can be no excuse; but for many people, whether male or female, anything up to two and a half inches of elevation may be of vital necessity to comfort.

*Heel-wedges.*—While on the question of heels it might be opportune to say one word about the wedging of a heel though I realize that this is a question of abnormal foot-gear rather than of the normal. In the first place I want to demonstrate to you the contrast between applying the wedge to the bottom of the heel and to the top. A glance at the line drawings below will illustrate the point adequately. Under no circumstances should any elevation be made on the inner side unless the heel is thrown out to the outer side at the same time, so as to ensure that the centre of gravity of the body still falls through the centre of the heel of the shoe (fig. 5).

This leads on to the consideration of whether the sole should be wedged on the inner side or not. It seems to me that this prescription is often given without

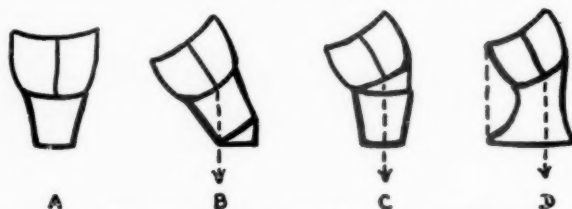


FIG. 5.—To show how the heel should be wedged on the inner side. The figures show the left heel seen from behind: (A) An ordinary heel of the "peg-top" type; (B) To show the effect of placing the wedge near the ground; (C) To show the effect of a corresponding wedge placed near the top of the heel; (D) To show how the lower part of the heel should be thrown out to the outer side to counteract the tendency for the foot to turn over to the outer side.

(From Mennell's "Physical Treatment". With acknowledgment to Messrs. J. & A. Churchill, Ltd.)

adequate consideration of the mechanical effect that it must produce. The longitudinal arch of the foot on the inner side is formed by the os calcis, the astragalus, scaphoid and first metatarsal. Anything that prevents the head of the first metatarsal from dropping downwards must of necessity tend to flatten the arch. On the other hand, if the heel is wedged upon the inner side and the sole is not raised, the tendency can only be to increase the arch and so relieve the structures in the sole of strain. It seems, therefore, that if we are to use the wedging of the sole aright, we should use it only for those cases in which restoration of mobility is impractical owing to pathological changes within the joints. To apply a sole wedge when we hope to maintain or to restore full mobility is calculated to defeat our main object in treatment.

Returning once more to the heel of the shoe, the "set" at which it is built is often of vital importance. The practice of turning inwards the angle of the heel at the forepart on the inner side is to invite the individual to evert the foot at every step that is taken. No last should ever be chosen in which the angle fails to touch the straight line drawn from the inner side of the heel to the inner side of the sole.

*Fitting.*—We now come to the consideration of the general type of shoe to be chosen by any individual. Attention has already been drawn to the danger of relying for selection of length on the measurement of the foot while sitting. It should be an invariable practice to measure it standing as well, as even if the length of the foot increases by only a length on standing, due allowance should be made. We then add the two boot-sizes necessary for the sliding forward of the front of the foot and finally any addition that may be necessary to allow for the shaping in front. We must add at least half a size to allow for a thin sock and the thickness of the shoe material. The question of breadth is as important as the question of length and again it is necessary to take the breadth standing and sitting. If a tape is placed around the foot at the level of the heads of the metatarsals, and the measurement is taken in the sitting and the standing positions consecutively, it is not at all an uncommon thing to find there is a difference in the two measurements of anything up to three-quarters of an inch (fig. 3). As, however, we have to supply a shoe of a length considerably in excess of the length of the foot, there is no necessity with a normal foot to do the same with regard to breadth. In fact if we do, it often leads to disaster and the shoe chosen is merely a sack. With one exception it is wise to choose from the point of view of breadth a shoe which is a quarter of an inch under the measurement of the breadth of the foot when standing. The one exception is when the patient is the victim of a hallux valgus with a bunion, and in this event the full measurement should be allowed and indeed something extra may be required.

*The upper.*—We now come to the consideration of the "upper" and here I would remind you not to lay too much faith in the statement of size placed on any shoes in the ladies' department. It is by no means an uncommon event to find a shoe that is labelled five actually measures seven or even more when examined with the shoe-stick. Unfortunately there seems to be no general law in the trade governing the length which a shoe is stated to be in the ladies' department. In the men's department the variation between the actual and the stated lengths is comparatively insignificant.

The shaping of the "upper" varies enormously according to the different lasts. In American shoes the depth of the front of the toe is usually maintained to the end of the shoe. In the eye of the English manufacturer this is not satisfactory, and it is customary to slope the front part of the shoe down gradually for a considerable distance. It is perfectly plain therefore that this practice must render the front part of a shoe so shallow that the big toe and second toe cannot find their way under this sloping roof. This extra length therefore is sheer waste. It is a particularly sad reflection that this fault is carried to extremes in the children's departments, and, wherever this slope is observed, due allowance must be made for it in estimating the effective length of the shoe.

For most of us a heel elevation of some sort is far more conducive to comfort than no elevation at all. Not long ago an attempt was made to popularize the heel-less shoe: it has not proved to be a success. If, however, we raise the height of the heel at all, the upper surface of the sole of the shoe must of necessity be on an inclined plane, and therefore there will be a tendency for the foot to slip forward down this inclined plane, thus tending to allow the heel of the foot to slip away from the heel of the shoe into the forepart. When this is allowed to take place it is obvious that we must be depriving the patient of the advantage of the extra length for which we have made provision in front. It is essential therefore for comfort that something should be provided to keep the heel back in the heel of the shoe. A narrow strap fixed with a button is totally inadequate and nothing less than two narrow straps fastened with buckles is in any way effective. This of necessity implies the instantaneous condemnation of the court or pump shoe for walking purposes. It is of course a matter of indifference what type of shoe is chosen if it is only to be worn while sitting at the dinner- or bridge-table; we are concerned to-day only

with the consideration of foot-gear that will enable us to walk or to stand about without risk of injury. If no adequate provision is made to keep the heel of the foot back in the heel of the shoe, it is obvious that something must prevent the foot from sliding down the slope provided by the inclined plane, and the only thing which can prevent it is the meeting of the front part of the foot with an opposition from the front part of the shoe. This suffices to prevent any forward gliding whatsoever of the toes; but if this happens, i.e. if the toes run up against the front of the shoe, nothing can prevent them from bending at the proximal interphalangeal joints. This will involve dorsiflexion of the proximal phalanges at the metatarsophalangeal joints and this involves an undue exposure of the heads of the bones in the sole. There is no allowance made in the fore-part of any shoe for flexion to any marked extent of the interphalangeal joints, and, if it occurs unduly, corns over these joints and callosities under the sole are almost inevitable.

Just as we have spoken of the tendency of the English shoemaker to slope the front of the toe from above downwards to an undue extent, so too is the tendency observed to do the same thing on the outer side—again especially in the children's department. It not infrequently happens that a shoe, admirable in every other respect, possesses this fault, with the result that there is no adequate accommodation for the little toe. The further result is that a painful corn in this position is a frequent occurrence. This fault is also one of the common causes of soft corns between the toes.

Often enough, and particularly when a lace-up shoe is selected, nothing can be found which will allow sufficient control of the heel of the foot in the heel of the shoe. A shallow pad of adhesive felt placed under the tongue will often solve the problem. Another very common fault in the designing of a shoe in this country, though this applies less to the American-made shoe, is the balance between the breadth of the heel at the back and the breadth at the level of the metatarsal heads. Very often the disproportion between the shape of our foot and the shoe that is offered us is absurd; but many of us require a relatively very narrow heel combined with a relatively broad front, a pattern which is often unobtainable from stock.

Finally, of course, when trying to select a pair of shoes it is necessary to measure both feet, as few of us are entirely symmetrical on both sides. In this event it is always wise to select shoes fitted to the larger foot and to make any necessary adjustment for the smaller one by wearing a cork in-sole or sock and a pad behind the supporting strap or tongue. Some retailers are willing to sell one shoe from one box and the other from another box, but it is not a common courtesy. It is also worthy of comment that to wear too short a sock or stocking is just as baneful as to wear too short a shoe.

#### CONCLUSION

The human foot is a perfect miracle of mechanical ingenuity and in the vast majority of people it should be perfect throughout life. With the exception of the victims of congenital abnormality or of gross disease or injury, a painless and perfect foot should be our heritage. Of this heritage we have been deprived by our foot-gear and I cannot help feeling that this evil thing is a reflection on the lack of co-operation between the shoe manufacturers, the anatomist and the medical man. The extent of the evil may be gauged by the extent to which chiropody has become a necessity in our civilized community; and the enormous variety of foot supports and foot-easers (often misappropriately so-called) which are to be found in every town and almost every village throughout the country bear an eloquent testimony to our foolishness in submitting to a tyranny, which is the direct cause of intense suffering in the feet, which is fatal for the all-important maintenance of a correct posture and which, most important of all, leads to irritability and bad temper.

## Section of Psychiatry

President—F. L. GOLLA, O.B.E., M.D.

(November 14, 1939)

### SIGMUND FREUD

Born in Freyberg, Moravia, May 6, 1856. Died in London, September 23, 1939

(Honorary Fellow of the Royal Society of Medicine)

**Dr. Ernest Jones:** I can best introduce what I have to say on the occasion of this memorial meeting by following something of the train of thought which I know Freud himself would have started in reflecting on such an occurrence. This memorial meeting has the appearance of a graceful act expressing, as it does, a tribute of gratitude to the man who has given something of value to psychiatry. Personally I take it that this is its essential meaning. Yet Freud was a man who was never content to regard the appearance alone of any human activity and who was prone to inspect its inner meaning and the motives associated with it before he would allow himself to form a final estimate of its significance. His mind constantly displayed an attitude of somewhat disconcerting scepticism. And he once remarked that the bestowal of honour on the living and the erection of monuments to the dead were often dictated by other motives as well as the manifest ones. He thought, in fact, that motives of affection and respect could be accompanied by less friendly and unavowed ones which were the expression of the wish to dispose of the personage concerned—as much as to say: “Now we have put you on a pedestal, so please be quiet and do not disturb us any further. We have shown our respect for you. What more do you ask of us?”

The world has rather naturally not welcomed such an attitude, which so often reveals aspects of mental life it would prefer to keep decently hidden out of sight. It tends to form an unfavourable opinion of men endowed with such attitudes and to retaliate by inspecting in its turn the source of them. There is no doubt that critical scepticism may have more than one origin. It may come from a sadistic pleasure in hurting other people by pointing out their weaknesses, and this would, I think, be a frequent judgement. I have the best of reasons, however, for knowing that with Freud, as with many other great men, it sprang from a different source. Freud was a man with a deep instinctive love for what may be called the positive in life, for the genuine. For him good feeling and truthfulness were one and the same. He was therefore especially interested in whatever interfered with the genuine and positive and was concerned to unravel the nature of any negative interferences, in the faith that then the goodness would be freer to act. “Debunking”—to use the present-day colloquialism—is an activity that may be impelled by love as well as by hate, that may indicate the philanthropist as well as the misanthropist. This is also the reason why those who rated Freud as a pessimist were wrong in their judgement. His disposition was unfailingly cheerful, and what they took for pessimism was actually an unusually stark realism, an exceptionally firm grasp of reality and dislike of illusion.

Freud himself was often the victim of insincere manifestations of respect, and indeed occasionally in forms so gross as to compel attention. The word “genius”,

for example, was often exploited in this fashion. I could quote to you extreme examples of its misuse. "Freud is a great genius, but only as an artist, not as a scientist"—a singularly inapt remark, since Freud happened to have very little sense of form in art, with the one exception of prose writing. Again, "Freud is a great genius, but as a philosopher, not as a scientist", also a poor judgement, since he had in fact a distinct aversion to philosophy and the philosophic way of thinking. Occasionally the full meaning of the phrase became so clear that one could not unfairly translate it with "Freud is a great genius, but he has such an uncontrolled imagination that most of his observations and conclusions are incorrect". In such cases one is impelled to ask what is left of the conception of genius after it has been emptied of all its content.

We are perhaps on safer ground when we speak of Freud as a great man. For there we can objectively point to certain qualities in him of the kind generally called great, which are also independent of the validity of his scientific work. In other words, even his opponents would probably call him a great man, because he possessed in an extraordinary degree a number of admirable virtues. Foremost among them was his exceptional honesty of mind and his devotion to the ideal of truth. He was always the first to point out the imperfections and misapprehensions in his work and in correcting them in the interest of greater accuracy he was indifferent to the charges brought against him of self-contradiction or changeability. His sensitiveness to the inner voice of criticism, however, was accompanied by a remarkable resistance to any outside influence or pressure. Here he displayed a courage in the face of bitter opposition, and a tenacity in his adherence to his hardly-won convictions, that in themselves raise him far above most of his contemporaries.

Freud was by nature a man of peace who attached more value to peaceful than to militant pursuits. Once only, at the outset of his career, did he enter into controversy with an opponent. All other attacks on his work—and no man could have had more—he answered in the same fashion as our great Darwin, whom he resembled in many other respects: namely, by simply producing a further piece of research. This attitude he adopted both from conviction and by temperament. He had little belief in the value of controversy in scientific matters. He observed how many other factors played a part in it besides the ostensible search for truth, such as the desire to prove oneself personally in the right, to score off an opponent, and so on. So he refrained from polemics as being something that wasted time and emotional energy and brought one no nearer to the goal. The greatness of his character thus showed itself both in his scientific work and in his attitude towards it. He was really serious in his endeavour to enlarge our sphere of definite knowledge.

I will venture to define a genius in science as someone who displays significant originality, and when judged by this criterion it would be hard to deny the title of genius to Freud. By significant I mean important, not merely a trivial novelty, and there can be little doubt that if Freud's ideas are true they are of fundamental importance. I need only refer to his central discovery of the unconscious mind, the discovery that what we had previously thought was the whole mind, i.e. consciousness, is a product of deeper layers and is constantly influenced by the sources from which it springs. And by originality I mean something more than flashes of insight, however brilliant and accurate these may be. With most of Freud's discoveries it is possible to point to precursors—indeed he himself called attention to them—who had made what may be called lucky guesses. Freud's merit lay in taking his new ideas seriously, in following them up in detail with unsparring labour, and in not resting until he had established them on a wide basis of correlation with other, known ideas. He once compared the difference in these two attitudes to that between a casual flirtation and a responsible marriage.

It has been well observed that most important discoveries in science are due to the invention of some method or technique, and Freud's were no exception to

this rule. All his psychological work really rests on his appreciation of what is somewhat misleadingly called "Free Association", and the method of research thus devised is the essence of psycho-analysis. With a ruthless determinism he took literally the maxim that contiguity in mental processes signifies a relationship in their content, an act of sheer originality that has had revolutionary consequences in psychology and psychiatry.

If we think of psychiatry in the literal meaning of the word, i.e. mental therapy, then there would be general agreement that Freud contributed more to psychiatry than any other single worker, since practically all psychotherapeutic measures now used are informed by his analytic approach. That only a proportion of such therapists take the trouble to learn the necessary technique for the proper handling of the analytic method does not alter the fact that they nearly all make some attempt to use it and find the conceptions introduced by Freud, such as repression, wish-fulfilment, &c., indispensable to their work.

Psycho-analysts themselves would not, I think, rate this psychiatric contribution of Freud's, grateful as it may be to suffering patients, as having the same value as his contributions to psychopathology and to psychology. I remember once asking him, long ago, which part of his work he himself estimated most highly. He said that the two books of his he liked best were the *Three Essays on the Theory of Sexuality* and the *Interpretation of Dreams*. Then he added, with a shrug of the shoulders, "but the conclusions in both are so obvious that I hope they will soon become antiquated". It is interesting to observe that this hope shows more signs of being fulfilled with the former book than with the latter. In spite of the tremendous storm of vituperation with which the conception of infantile sexuality was greeted it has nevertheless during the past thirty years won a very widespread acceptance. The number of people who are shocked by sexual manifestations in childhood, and who are impelled to explain them away as an abnormal precocity, must be getting fewer every year, and the indirect effects of the book in social and pedagogic fields are, I think, unmistakable.

On the other hand there are, in my opinion, very few who have been able fully to grasp the implications, or even the meaning, of Freud's theory of dreams; even analysts themselves often show an imperfect apprehension of it. The book is a very difficult one and so is its thesis, but this is not the only reason for the relatively slight influence it has exerted. A deeper one is that it attempts to make us familiar with a mode of thought that is extremely repellent to our conscious minds, namely that of the unconscious. This mode of thought, as had been dimly discerned by a number of great men, including notably Hughlings Jackson in this country, bears an extremely close similarity to that of the insane. To recognize this in oneself, and still more to feel it as an intimate part of reality, is a feat of which not many find themselves capable. This is one reason, among others, why psycho-analysis has been able to advance more extensively in the psychopathology of the neuroses than in that of the psychoses. With the former it has reached very far into the ultimate problems of ætiology itself, whereas with the latter its contribution to ætiology has been of a much more indirect, though none the less important, kind. Freud's brilliant analyses of the mental mechanisms in paranoia and melancholia, and the interpretation of schizophrenic symptoms his work has rendered possible, have given an entirely fresh insight into the meaning of psychotic processes. Extending into this field the knowledge he had gained in that of the psychology of the neuroses, he was able to demonstrate two important things concerning the mental manifestations of insanity. The first was that they had a meaning. The view previously held of them was that they were a meaningless jangle resulting from damage that had disordered the organ of thought, i.e. the brain. *A priori* this would be a perfectly plausible explanation of them, but his researches have shown that it could not be the true one. He found that, just as with the neuroses, a meaning and purpose could be

discerned in psychotic symptoms, in both the prominent and the trivial ones, and he provided a methodological approach by means of which it is open to everyone to test this conclusion. The second thing was this. He showed that psychotic manifestations were for the great part of a secondary nature, being in effect complicated defences against some more fundamental difficulty or attempts to deal with this. Delusions, hallucinations, depressions, and many other of the startling phenomena that distinguish the insane from the sane are thus seen not to constitute the essence of insanity—even in the purely mental field. This discovery has had the effect of clearing the way, of so to speak penetrating beyond a mass of problems that are now seen to be of a purely secondary nature, however interesting in themselves. In this Freud would appear to have rejoined the neurologists since these have always taken the view that the phenomena in question are secondary. But whereas they simply brushed them aside, Freud, as I say, worked steadily through them and in doing so learned much of importance concerning the more fundamental matters to which they are a reaction. He came to see that these have to do with the primordial problem of the relation of the ego to reality, and his later researches carried him far into this obscure field and into the burning question of the essence of insanity.

From a human point of view it is interesting to note that Freud did more than any other psychiatrist to forge links between the normal and the insane mind, to show that the differences between them are quantitative rather than qualitative. Just as he discovered that the neurotic suffered from the effect of conflicts that are not peculiar to him but are the common lot of mankind, although dealt with otherwise by the so-called normal, so he was able to show that the apparently inconsequent, illogical, and unintelligible mental processes of the psychotic patient are not peculiar to him but find their counterpart in a deeply buried layer of every human mind. This unconscious mental stratum, or pre-logical layer, cannot properly be given the name of psychotic, because it may or may not undergo a psychotic development later, but the mental processes comprising it follow extremely similar laws to those of psychotic thought and display all the extraordinary features that make this so extremely alien to our ordinary conscious thinking.

This forging of links between apparently disconnected processes was highly characteristic of Freud's work. His researches reached into many spheres of mental activity: psychopathological symptoms, the meaning of dreams, the unconscious processes underlying wit, the genesis of character formation, the study of artistic creativeness, of myths and folklore, of religious ritual and belief—to mention only some of them. They even extended to the remote past of man where various data permitted him to draw analogies between the development of the mind in the individual and that in the race. In all these fields he established innumerable correlations and sought constantly for general laws that would co-ordinate his mass of observational data. I will not touch on the peculiar mental mechanisms he exposed in these disparate phenomena, but would stress the connexions he established in all of them with what he came to regard as the main clue to the understanding of the mind, namely, the fundamental importance of the conflicts between instincts in the earliest situation of life where the infant is concerned with its first human environment, its parents. He thus introduced into, amongst other fields, that of psychiatry, a biological and genetic point of view that must surely be of increasing importance as our knowledge deepens.

Freud was a remarkable example of a natural psychologist. By this I mean that he had the rare faculty of recognizing a psychological fact and of respecting it instead of discounting it. One example will illustrate this faculty. In the earliest days of his work he collected a number of cases where sexual seduction of a child by an adult appeared to play a part in the genesis of the later neurotic symptoms. Then he found that several of these stories were simply untrue—there had been no seduction. Most workers would at this point, I am sure, have shrunk back from the morass

in which they found themselves. Not so Freud. He held fast to the fact that the patient had told him these stories. This remained a fact, and he turned his interest to it, with the result that he discovered the importance of the infantile phantasy life in the genesis of the neuroses.

Freud will for two reasons always remain a memorable figure in the history of science. First for the fact that he conquered a new province for science, that of the unconscious mind, with all the associations that go with this of methodological technique and the biologically genetic approach to the mind. Then for the example he has given us of a pioneering investigator, with all the endless patience in observation, freshness of insight and daring readiness to face the unknown, that characterize the great pioneer. Let me stress above all the last of these qualities. We know that physical courage is—fortunately in these times—a common enough human virtue, that moral courage is rarer, and that rarest of all is intellectual courage. I do not think we know of many figures in the history of the world who have equalled Freud in this rare and transcendent quality.

**Professor Millais Culpin :** In assessing Freud's place in the history of medicine the historian would have to study a gradual change in mental outlook rather than a clear-cut acceptance of new theory and technique such as characterized the triumph of the views of Lister. There are no striking landmarks to indicate this change, and it is indeed probable that the newer generation of our profession already regards many of Freud's conceptions as part of the general heritage of medicine and is unaware that we owe them to the genius and the labour and courage of one man.

There has been, in the history of medicine, an ever-recurrent controversy between one school of thought which would claim to find the final explanations of bodily processes and, indeed, of human behaviour, in mechanical terms, and another which demanded recognition of some unifying or even governing principle by which the living organism acts primarily as a whole. As an example of such a final explanation one may cite the *nervus force* of Sprengel (wrongly attributed by him to von Haller) or that application of Pavlov's work by which man's whole behaviour becomes but a series of conditioned reflexes; for the governing principle we may turn to the *phusis* of Hippocrates, the *anima* of Stahl, or the instincts of McDougall.

The wonderful growth of scientific knowledge, and of its application to medicine, that began in the latter half of the nineteenth century, swung the pendulum well over to the mechanistic view, and the Oxford English Dictionary expresses that view when it defines neurosis (not in the pathological sense) as "a change in the nerve cells of the brain prior to, and resulting in, psychic activity", and supports the definition by quotations from Huxley and Romanes. Our attitude towards some problems of clinical medicine was determined by such a metaphysical conception, and little search is needed to find examples. We read as late as 1916, for example concerning hysterical aphonia :—

The abrogation of function in such cases was due, not to the existence of any organic lesion, but to a temporary suspension of neuron impulses from the higher cortical cells of the central nervous system to the periphery—in other words, to a sudden arrest, probably of vasomotor origin, of those volitional impulses requisite to produce speech.

Such an explanation, which represents quite fairly the authoritative view of that time, stands at the opposite pole from that which regards the symptom as a problem of behaviour behind which there stand mental processes of which the patient is unaware—a simple enough pathology that would meet with but little contradiction to-day. It would be wrong to assume that such a pathology is new; it might perhaps be found in the writings of the ancients, but we owe to Freud the establishment of the principles that enable us to accept calmly the fundamental change of outlook illustrated by this apparently trivial example.

With the change of outlook has come an unwitting absorption of psycho-analytical ideas and terminology. The theory of unconscious ideation is an old one, but when

Freud brought it out of the realms of philosophical speculation and proved its necessity for the understanding of human behaviour in both the apparently healthy and the afflicted, he shook our egocentric pride to a degree that can be estimated on reading the late Charles Mercier's once famous article in the *British Medical Journal*, of which it was often claimed that "Thanks to Charles Mercier, psycho-analysis is now dead". Mention of the unconscious now passes as a commonplace, as do references to some of the more specific factors concerned in unconscious mental processes. We are all prepared to recognize inner conflict as a cause of psychoneurotic difficulties and, whether or not he is able to give it, the medical practitioner will usually admit the need for attention to the emotional difficulties of his patients. The effect that the early emotional environment of the child has upon his reactions in later life is well known, even though the development of the super-ego has not yet become a part of our general knowledge. In short, the influence of Freud's discoveries has profoundly altered our outlook upon the pathology of the commoner mental disorders.

In the period when a complete reliance upon test tubes and microscope slides marked one of the dark ages of psychology in medicine, there was an uneasy feeling that all was not well, shown by regrets at the disappearance of the old family practitioner who "knew the patient's constitution". What the phrase seemed to mean was to know him as a human being, with desires and emotions, perhaps with queer nervous symptoms; and now and again we read an article in which the writer gives, out of his own experience, some advice, often useful and wise, on that facet of practice. In such an article Freudian conceptions and terminology may be freely used but, in the absence of direct mention, it may be suspected that the writer is unaware of the source of his knowledge; sometimes, indeed, a slighting reference to Freud makes the suspicion a certainty.

One phrase introduced by Freud has met a curious fate. That is "anxiety neurosis", coined by him to indicate a condition, separated by him from the rubbish-heap of "neurasthenia" in 1895, that he considered due to sexual stimulation without gratification. This, he said, was a true neurosis—using the word in its correct physiological sense—which belonged to ordinary medicine and offered nothing to psycho-analysis. The phrase has now become popularized, its original meaning is lost, and it seems likely to become as wide in its applications as was the now nearly obsolete "neurasthenia".

In our attitude to the emotional, as opposed to the physical or anatomical, aspect of sex there has been as radical a change as in the more obviously clinical matters. It is noteworthy that the intellectual giants of the nineteenth century, often compelled to fight against obscurantism in its many forms, accepted and applied without question the current taboos upon sex. These taboos have been so often held up to ridicule that we fail to appreciate the strength they possessed. The late Dr. G. E. Herman, however, in his textbook on diseases of women, confesses with delightful frankness both his acceptance of the taboo and its power over our own profession where, both in his 1899 and 1907 editions, we read "Hence anomalies of sexual feeling are probably commoner in women than in men. The reserve which properly attends this subject prevents scientific investigation of it".

Another passage runs: "Dilatation of the cervix . . . cures dysmenorrhœa, establishes sexual desire and pleasure, and sometimes cures sterility. I cannot tell how often this happens, because the subject is not one upon which routine inquiry can be made." There is some obscurity in the statement, and a modern reader might wonder why routine inquiry could not be made about sterility, but to the generation for which Herman was writing there was no doubt as to his meaning; to them it was beyond question that sexual desire and pleasure were subjects unmentionable even in the consulting room of the gynæcologist.

It is interesting to speculate whether this sentiment, based upon the taboo, really existed in any strength on the part of the patient. It is almost certain that

the manifold miseries that we now know to exist because of emotional difficulties in the sex life would, even in those days, have been freely discussed with any practitioner whose own inhibitions did not check the confidences. When the descriptive writings of Havelock Ellis first appeared they seemed to belong to a world different from that in which the ordinary doctor moved, and there are still some of us to whom that world has no existence within our own experience, and to whom it never will have existence; but there remain few who would accept without question that the scientific investigation of some of the most distressing troubles of our fellow beings should be prohibited because of a taboo which, occurring in any other race than our own, would long ago have become itself an object of curiosity and research.

The changes in our ways of thinking here considered have had in them an element of the destructive, a clearing away of obstacles; and for such a process there was need. The positive side of Freud's influence is obvious to us all. With a recognition of the nature of psychoneurotic disorders—a problem that had puzzled us from the time of Galen—has come an interest which was previously impossible. We see a wide movement for ensuring treatment for the sufferers, and a recognition of the need for teaching clinical psychology in our curriculum. It is not necessary to emphasize this. What we can and should recognize is the change of attitude that has come about in all of us to make it possible.

**Sir Walter Langdon-Brown:** I must crave your indulgence because I have to appear as Professor Mapother's substitute at the shortest possible notice. There is, however, perhaps something to be said from the point of view of the general physician who is not a professed adherent.

In the first place Freud provided us with a new mental apparatus, as new in our time as Bacon's Inductive Method was in his. How new it is can be gathered by comparing Locke's admirable essay on the Human Understanding with any present-day textbook of psychology—Locke's study was one of the surface anatomy of the mind with no reference to its hidden depths.

The truth of a theory or alleged discovery is often proved by the way its influence spreads into other departments of thought or activity. Darwin's work is of course an outstanding example of this.

The stages are usually these—first a period of fierce controversy in which all the conservative tendencies offer a stubborn resistance—and then an infiltration of the new idea into unexpected fields of thought. While the old guard is resisting a frontal attack they find that their wings have been turned. The controversial stage for Darwin was almost as long as for Copernicus. As far as I know the first overflow appeared in Walter Bagehot's curiously named "Physics and Politics" wherein he used the term physics in its oldest connotation of *φύσις*, i.e. Nature. After that the spread was rapid.

Let me recall something of what I recollect of the pre-Freudian scene. In the nineties of last century Frazer's "Golden Bough" (and later his "Folk Lore in the Old Testament") was the herald of an approaching change. Verrall's study of Euripides infuriated the orthodox classical scholars who were unwilling to see in him the shrewd critic of accepted things. Haddon's expedition to the Torres Straits converted certain physiologists into psychologists, Maudsley's approach to mental diseases was breaking new ground as was Hughlings Jackson's conception of progressive dissolution of the layers of the mind. I well remember the interest aroused by the publication of Havelock Ellis's "Man and Woman". Meanwhile the ancient art of dream interpretation had sunk into mere chap-books for the illiterate.

Looking back it seems to me that except for the last detail the ground was being prepared by studies of mythology, anthropology of the ancients and of savages alike,

and of mental disease. But there was no real co-ordination between the various themes that were to be heard. When Freud took up the conductor's baton, as it were, this preliminary tuning-up assumed symphonic structure. The result, as we know, was by no means to everyone's taste, and the stage of active controversy is hardly over. But the infiltration into other fields of thought—to return to my original metaphor—began very quickly and it now tinges everything to a greater or less extent. It is significant that one of the earliest and, I think in some ways, still the best interpretation of Freud for the ordinary reader, is from the pen of a botanist, Professor Tansley, showing its biological appeal.

As individuals we have naturally tended to rank preservation of the individual as the primary and most deep-seated impulse, although all history resounds with self-denying examples to the contrary. Biologists have again and again pointed out that survival of the species ranks far higher with Nature than perfectibility. She is content with imperfect machinery if it has sufficient survival value. Hence the burdens from the past we have still to carry and the urge to the creation of new life. Professor Danforth recently remarked "sex started as a difference between germ cells and has evolved into a difference between individuals". Herein, stated in most general and superficial terms, is the biological basis of what I take to be the main doctrine.

It is, however, important to remember in view of the criticism that the superstructure is chiefly composed of airy imaginings that Freud's earlier professional life was devoted to profound neurological studies—anatomical, histological, and pathological, and it is not likely that such studies failed to make a deep impression and to provide a solid foundation on which he built a remarkably unifying conception of life.

I turn to another aspect of this, our memorial tribute to Freud. If it be true that "Blessed are the persecuted", he was thrice blest. It sounds paradoxical to say that in earlier days he was both persecuted and neglected. It was a great surprise to Vienna to find that the most notable Viennese name outside Austria was Freud's. It is not so surprising that hostility should have been aroused by what appeared to be the most challenging and aggressive way of stating his views. I do not think that he tried to attract attention by being deliberately offensive to others—that would have been out of keeping with the rest of his character. He stated the truth as he saw it, and he would not compromise with that.

I am thinking also of his final persecution. Quite recently I listened in to the Trial Scene of St. Joan. It is much more poignant now than when it was written in 1920. Then it was a grim bit of past history of sadistic tyranny over liberty of thought; now it has become again almost unbearably topical. All the old mediæval methods have been revived with the added refinements of modern science. But ceremonial burning of Freud's books cannot prevent the spirit therein contained from rising phoenix-like from their ashes. We had taken liberty of thought so much for granted that it took us some time to realize that the price of liberty is still perpetual vigilance. The monstrous gods of old are hammering at the door of civilization again and Wotan, himself doubly forsworn, becomes the tutelary deity of all who break faith. Full of years and broken in health Freud would bow the knee to no false gods. The grim and disgraceful scene of the raiding of his home and the robbery of his money is lit up by his philosophical humour for it is said he remarked, "I have never charged so much for a single visit".

After storm, calm. It is pleasant to know that in this country he found a peaceful haven. Of the end I will speak in Bunyan's words:—

"When Mr. Standfast had set things in order, and the time being come for him to haste him away; he also went down to the River. Now there was a great calm at that time in the River."

## Section of Anæsthetics

President—HAROLD SINGTON, M.D.

[December 1, 1939]

### Status Lymphaticus

By JOHN F. TAYLOR, M.D.

STATUS LYMPHATICUS, to use its most popular name, is also known as status thymico-lymphaticus, or lymphatism. It is alleged to predispose to sudden death from trivial causes, and most commonly perhaps, during or shortly after anæsthesia. Such trivial causes include comparatively light blows and slight injuries, plunging into cold water, the injection of antisera, vaccines, drugs, and local anæsthetics. Chloroform was the first inhalation anæsthetic considered dangerous, but deaths attributed to status lymphaticus have now occurred, I suppose, with almost every anæsthetic known, down to nitrous oxide.

Apparently the first relevant observation was made by Felix Platter, as long ago as 1641. Platter recorded three cases of sudden and unexpected death in one family, in children with large thymus glands. For more than two hundred years, similar cases were noted at intervals, and though interest in the large thymus seems to have waxed and waned, it received a fresh fillip in 1829 when Kopp attributed "laryngo-spasm" or "laryngismus stridulus", to the pressure of an enlarged thymus. This condition characterized by "crowing" breathing, cyanosis, dyspnoea, sometimes convulsions, and possibly death, is now often spoken of as "thymic asthma". It was this long-standing belief in the responsibility of the thymus which has caused so much misunderstanding. The term "thymic death" appeared, and the size or so-called persistence of the thymus was looked upon as the essential and possibly the only abnormality needed to explain these cases. This is far from being the present conception of the morbid picture.

Friedleben in 1858 disputed the possibility of the pressure of the thymus causing laryngo-spasm, and took the view moreover that many so-called enlarged thymuses were within normal variations in size. In 1889 and 1890, Paltauf recorded a number of sudden deaths, in adults, with a *general* enlargement of lymphoid structures, including the tonsils, lingual tonsil, lymphatic glands, spleen, and a thymus which varied in size. He found also a narrowing of the aorta. He agreed with Friedleben that so-called "thymic deaths" were not due to the pressure of the thymus, but considered they were due to a general lowering of resistance, dependent on a constitutional anomaly of "lymphatic-chlorotic" type, and the anatomical changes were only the gross manifestations of nutritional defect which he called "status lymphaticus". This description and general conception of Paltauf form the *basis* of the views of modern believers.

In 1895 Kundrat recorded 10 cases of death under or immediately after anæsthesia with chloroform, or a mixture containing chloroform. He noted that the heart failed before the respiration, and that post mortem it was flaccid and dilated, particularly as regarded the right ventricle. In addition he noted as well as the

enlarged lymphatic tissues recorded by Paltauf, swelling of the solitary follicles, and Peyer's patches in the intestine. He also called attention to the presence of a persistent and enlarged thymus in Graves' disease.

Little has to be added to Kundrat's picture. It is said now that children with status lymphaticus are well nourished, but of pasty complexion, increased subcutaneous fat, limbs rounded and sleek, skin soft and smooth. Adults tend to have scanty axillary hair, and males show the female arrangement of pubic hair, and deficient hair on the chest.

Since the thymus still forms a part of the picture it may be well to say a few words about its morphology and behaviour. On this subject the researches of Hammar are outstanding. The thymus arises as two lobes, one from each end of the third branchial cleft, which eventually fuse in the centre. It starts as an epithelial structure, but early in foetal life becomes infiltrated with lymphocytes, and though it preserves a medulla and cortex until late middle life, it appears to function as lymphoid tissue. The involution or diminution in size does not take place as early as was formerly supposed. Hammar found that it grows larger until puberty, 11-15 years, and not till then does involution begin. He believes that it is a factor to be reckoned with throughout life, and that there is no longer any question of persistence of the thymus in the usual meaning of the term. The thymus diminishes in size in wasting diseases, and this must be borne in mind when estimating the normal weight. Considerable variations are shown at all ages.

The thymus is enlarged in Graves' disease, Addison's disease, acromegaly, and after castration, suggesting a relationship with the endocrine glands, though there is little evidence that it has itself any internal secretion.

In 1927 Greenwood and Woods, after a statistical investigation, launched an attack on the belief in status lymphaticus. They attributed the enthusiasm for Paltauf's theory to two causes :—

(1) It revived the doctrine of "crases" or "temperaments", which was older than Hippocrates, but developed by Galen. This doctrine had been more or less abandoned by the materialists of the first half of the nineteenth century. Now evidence of a "lymphatic-chlorotic temperament" could be "put on a plate" in the shape of thymus and lymph-glands, and the doctrine received new life.

(2) It restored the omniscience of the pathologist. Now he could always find the cause of death, not only in the post-mortem room, but also in the witness box. Greenwood and Woods maintain that there was no criterion by which the morbid picture of status lymphaticus could be identified. They quote figures showing that different observers must have very different standards of proof. In the last war they say, Groll found in healthy soldiers evidence of status lymphaticus in 56% of bodies, Benecke 15%, and Forni in less than 2%. Startling discrepancies indeed, but which figures should we accept?

Greenwood and Woods summarize thus: "At the present moment we have not sufficient knowledge of the frequency of the single and combined anomalies which have been thought to define anatomically status lymphaticus, or status thymico-lymphaticus in a healthy population, to permit us to assign the least importance to recognition of these stigmata in the bodies of those whose deaths apart from status lymphaticus would in more pious, but not more superstitious, days have been attributed to the visitation of God." Again they describe status thymico-lymphaticus as a good example of the growth of medical mythology in which a nucleus of truth is buried beneath a pile of intellectual rubbish, conjecture, bad observations, and rash generalizations.

In 1928, following Greenwood and Woods, Marine had published an article supporting the general idea of Paltauf. In his view lowered resistance is the outstanding known clinical manifestation of status lymphaticus in man, and experimental

status lymphaticus in animals. This lowering is not specific to any one stimulus, but is shown to a variety of drugs, to antisera, to vaccines, to anaesthetics, local and general, and possibly to the physical or psychical effect of the injecting needle. He thinks that status lymphaticus may be defined as a constitutional defect, usually congenital (though it may be acquired), dependent on an inadequacy of some function of the suprarenals, sex glands, and autonomic nervous system. This condition is anatomically characterized by delayed involution of the thymus, and the other stigmata commonly described. He calls attention to the large thymus found in Graves' disease, Addison's disease, and after castration, and the relationship of the thyroid, the adrenals, and the gonads.

In the meanwhile a committee appointed by the Medical Research Council, by collecting data from many parts of the country, were endeavouring :—

(1) To arrive at standards of the weight of the thymus in proportion to body-weight, sex, and age.

(2) To investigate closely the precise cause of death in persons dying suddenly from unexplained, or trivial causes, where the only abnormality found was the presence of a large thymus.

It is noteworthy that the thymus still appeared to be considered the crux of the question !

A great deal of material, not only thymuses, but other lymphoid tissue, was weighed and measured, and statistical analyses made by Young and Turnbull, who found their results in agreement with most previous observers. Twenty-three cases of deaths under anaesthesia were investigated. Young and Turnbull considered it impossible to attribute any of these to lymphatism because the effect of shock and anaesthetics cannot be estimated after death.

Their conclusion was : " In the opinion of the Committee the facts elicited in the present inquiry are in harmony with those of Hammar, 1926, and 1929, and Greenwood and Woods, 1927, in affording no evidence that so-called status thymico-lymphaticus has any existence as a pathological entity."

*The Lancet* then appeared to administer the death-blow in a leading article with the portentous title " The End of Status Lymphaticus ".

Yet the condition is still freely discussed in the medical press of many countries.

Campbell has written three provocative articles. He accepts Marine's definition, and considers the condition of much greater importance to the world in general than the rare deaths attributed to this cause would indicate, and stresses the susceptibility of its subjects to infection. He believes there are various grades of status lymphaticus, and is not impressed by the size of the thymus as an indication of its severity. He considers that lack of adrenal hormone produces the condition. In 1932 he discussed the causes of death in status lymphaticus, and after mentioning a number of theories including that of allergy, and of " lympho-toxins " produced by the excessive lymphoid tissue, he considers that only three theories are worthy of consideration :—

(1) The pressure of an enlarged thymus on the trachea or nerve structures of the mediastinum.

It seems difficult to believe that a relatively soft organ like the thymus can exert any effective pressure on a resistant structure like the trachea, yet Campbell quotes no less an authority than Chevalier Jackson as having seen through the bronchoscope narrowing of the trachea produced by an enlarged thymus in many cases with symptoms of wheezing, dyspnoea, and impending asphyxia. It seems easier to envisage pressure on the recurrent laryngeal or vagus nerves. In the case of the recurrent laryngeal, abductor paralysis might well cause the symptoms of laryngospasm, while the alarming and often fatal results of pressure on the vagus in the neck are well known.

Before we leave this pressure theory, I should like to refer to a short paper by Alan Moncrieff, on enlargement of the thymus in infants. Moncrieff describes a series of cases of attacks of syncope with dyspnoea, and cyanosis of the face. In these a greatly enlarged thymus was demonstrated by X-rays. Radium was applied over the thymus, and produced very marked reduction in its size with disappearance of the symptoms.

(2) The second theory discussed by Campbell is based on the general hypoplasia of blood-vessels said to be present in status lymphaticus, and it is suggested that sudden exertion or stress of induction of anaesthesia may rupture a weakened vessel in the brain. Very few cases seem to have been recorded to support this view.

(3) The third theory is based on the observation that in cases showing the picture of status lymphaticus, there is evidence of deficiency of the adrenals. The heart is already undernourished and rapidly exhausted, and when deprived of adrenal help, collapses. In a later paper, 1937, Campbell summarizes his views on status lymphaticus thus: "Those who have had personal experience with such cases, and those who have given the subject careful investigation and study, cannot doubt the existence of a pathological condition of such lowered resistance and hyper-susceptibility that the patient so affected is in danger of sudden death from trivial causes."

He again stresses the point that lack of the secretion of the adrenal gland, particularly of the cortical hormone, is the essential feature.

He makes suggestions as to the diagnosis of the condition in life:—

(1) The external peculiarities, such as pasty colour, increased subcutaneous fat, deficient bodily hair, and the rounded contour of the limbs.

(2) Hypertrophy of the thymus as shown by X-ray pictures which should be taken both laterally and antero-posteriorly.

(3) Thymic stridor with cyanosis of the face.

(4) The presence of enlarged tonsils, adenoids, and lingual tonsils. Possibly there may be palpable glands and spleen, but the general fatness may well hide this.

(5) Signs of cardiac degeneration, such as low blood-pressure.

(6) Allergic signs, such as hay fever, asthma, migraine, and urticaria, may be associated with the condition. Histamine susceptibility may be increased.

(7) Evidence of adrenal insufficiency including leucocytosis, hypotension, increased coagulation time, hypoglycaemia, and decreased alkaline reserve.

Campbell does not suggest that any of these signs are diagnostic, but that in combination they well give rise to suspicion and various lines of treatment may then be indicated. The general resistance may be improved by the administration of adrenal extract. If definite evidence of an enlarged thymus producing local symptoms is found, it may be reduced in size by X-ray, or radium therapy as shown by Moncrieff. The alkaline reserve may be maintained by carbohydrates and bicarbonate.

Before operations, physical and mental rest should be maintained for some days—if necessary with sedatives. It may be possible to test susceptibility to various anaesthetics by the administration of small doses, but the doubtful value of this procedure might well be outweighed by the alarm and perhaps annoyance of the patient. An injection of adrenalin may be given immediately before operation, and held in reserve for any signs of post-operative collapse.

In this brief record of the history of the problem I have tried to put before you the picture of status lymphaticus as it has been evolved, and some of the theories of those who have studied it. The solutions have ranged from simple trust in the large thymus to an intricate process now centred on the adrenals, with the anatomical changes taking secondary place. Some view it as a figment of a

disordered mind, an excuse for anæsthetists, or a cloak for incompetent pathologists. Others regard it as a very real and perhaps frequent danger.

From my own experience during the last six years I have selected 14 cases of unexpected deaths under anæsthesia. The patients seemed to be good anæsthetic risks, were not subjected to an operation which should entail shock, and post mortem showed no unsuspected disease to account for death. They all showed the general picture of lymphoid hyperplasia. The thymus varied in size, but in each case was definitely above the average for the age, according to the figures of Hammar. Of these 14 cases, 8 were male and 6 female. The youngest patient was two months, the eldest 7 years. The operations included tonsillectomy, circumcision, cleft palate and appendicectomy. It is noteworthy that four of these patients had passed through anæsthesia safely on previous occasions.

In all these cases the post-mortem examination showed acute congestion of the lungs with or without some degree of collapse, in fact the manifestations of shock. There was always present some degree of flaccidity and pallor of the heart muscle. In no case was there any evidence of complications, such as the inhalation of blood or vomit, of undue hæmorrhage, or any gross disease.

While some operations took as long as forty-five minutes, one death occurred in two minutes before the operation was started. Ether was used in every case, sometimes alone, sometimes with oxygen, eight times after ethyl chloride, and once with gas. Chloroform was never used. I have notes as to premedication in only four cases: three times atropine was used and once paraldehyde. The only common feature in these anæsthetics was ether.

I have omitted other cases of unexpected death under anæsthesia, in which an obvious, even if slight, toxic element was present, such as acute mastoiditis without extension, and an acute appendicitis of only twenty-four hours' duration. Strange as it may seem, I have not seen during the last six years any other death associated with anæsthesia without some obvious explanation. As Young and Turnbull have said, there is no criterion by which we could measure the effect of anæsthesia, or the degree of shock sustained in the dead body. I think it will be agreed that in the cases I have selected, there is no reason to think that either of these causes was *likely* to be sufficiently severe to bring about death. If these deaths are simply due to misfortune, in which one may include mismanagement of the anæsthetic, I can only feel that in my experience, patients with the lymphatic stigmata are very unlucky. Another impression I have is that the lymphatic child succumbs more readily to infection. Every year I see a number of young children who have died unexpectedly, and find evidence of bronchopneumonia. It is obviously impossible to estimate in cubic millimetres the volume of lung affected, or the number of milligrams of toxins in the body, but it has seemed to me that the lymphatic child dies more quickly and with smaller lesions than the non-lymphatic. This may be only "a rash generalization".

Of sudden death from other trivial causes, I will mention three:—

(1) A healthy boy of 7 was playing in the street, when he was bumped over, not run over, by a motor car. He was picked up at once, and found to be dead. At post-mortem the only injury found was a simple fracture of one rib without displacement. The general signs of status lymphaticus were present.

(2) A boy, aged 14. Well grown and muscular. Notoriously keen on running and boxing. One afternoon after running 220 yards in some sports he collapsed and died. He showed a rather flabby heart, and a fairly recent meal in the stomach. It was unwise of him, we may say, to run soon after a meal, but what boy does not do it? This one was unlucky, and he showed all the stigmata of lymphatism.

(3) (This is perhaps a border-line case.) A man, aged 25, well built and muscular, over six feet in height, had recently had a mild attack of influenza, but was apparently almost well

again. At any rate he was up and about and carrying on his normal life. In a mild altercation with a friend he received a blow in the chest and immediately collapsed and died. He showed some quite trivial bruises, the largest one over the heart I think might have made him feel sore for a few days, but no more than that. There were no other injuries. The heart was relatively rather small, its muscle flabby and pale, and on microscopical examination showed a very slight degree of fatty degeneration. The lungs were intensely congested. The thymus weighed 40 gm., which is definitely above the recognized average for the age of 25. The spleen was enlarged and there was general enlargement of lymphoid tissue throughout the body. It may be argued that the only explanation needed here is degeneration of the heart muscle following influenza, but this was so slight that I am inclined to think that the lymphatic condition must have been a factor in this unfortunate result.

Though these cases prove nothing, they are at least very suggestive.

A few weeks ago I wrote to three pathologists who perform many post-mortem examinations for coroners and asked them to give me their general views on status lymphaticus. Dr. Eric Gardner, while feeling sure that he has seen cases in which this condition is the predominant if not the only cause of sudden death, has not seen in recent years any anæsthetic death which he attributes to status lymphaticus. He thinks there are very often points in the administration of the anæsthetic which the pathologist is not in a position to know. Dr. Skene Keith has the definite impression that subjects with the stigmata of status lymphaticus die more readily than those without them. Sir Bernard Spilsbury regards status lymphaticus as a manifestation of alimentary toxæmia or bacteriæmia which produces an irritation overgrowth of lymphoid tissue. It would also explain the almost constant finding of fatty degeneration of the heart muscle. This renders the patient more susceptible to death from trivial causes, of which anæsthetics form one group. (He regards chloroform as particularly dangerous and attributes the reduction in child mortality from anæsthetics to the great diminution in the use of chloroform in recent years.) This, he says, is his conception of the condition, in which he is a "firm believer".

#### SUMMARY

For more than two hundred years sudden and unexpected death from trivial causes was thought to be explained by the enlargement or so-called "persistence" of the thymus. Nearly fifty years ago it was realized that the thymus was only one item in a general lymphoid hyperplasia, which was thought to be indicative of, but not the cause of, some constitutional anomaly. This condition was called status lymphaticus. This general conception seems to have been lost sight of in the repeated investigations made on the variations in size of the thymus. Attention has been drawn to the association of a large thymus with Addison's disease, Graves' disease, and deficiency of the sex organs. One theory advanced in recent times is based on a belief that adrenal insufficiency is the essential factor.

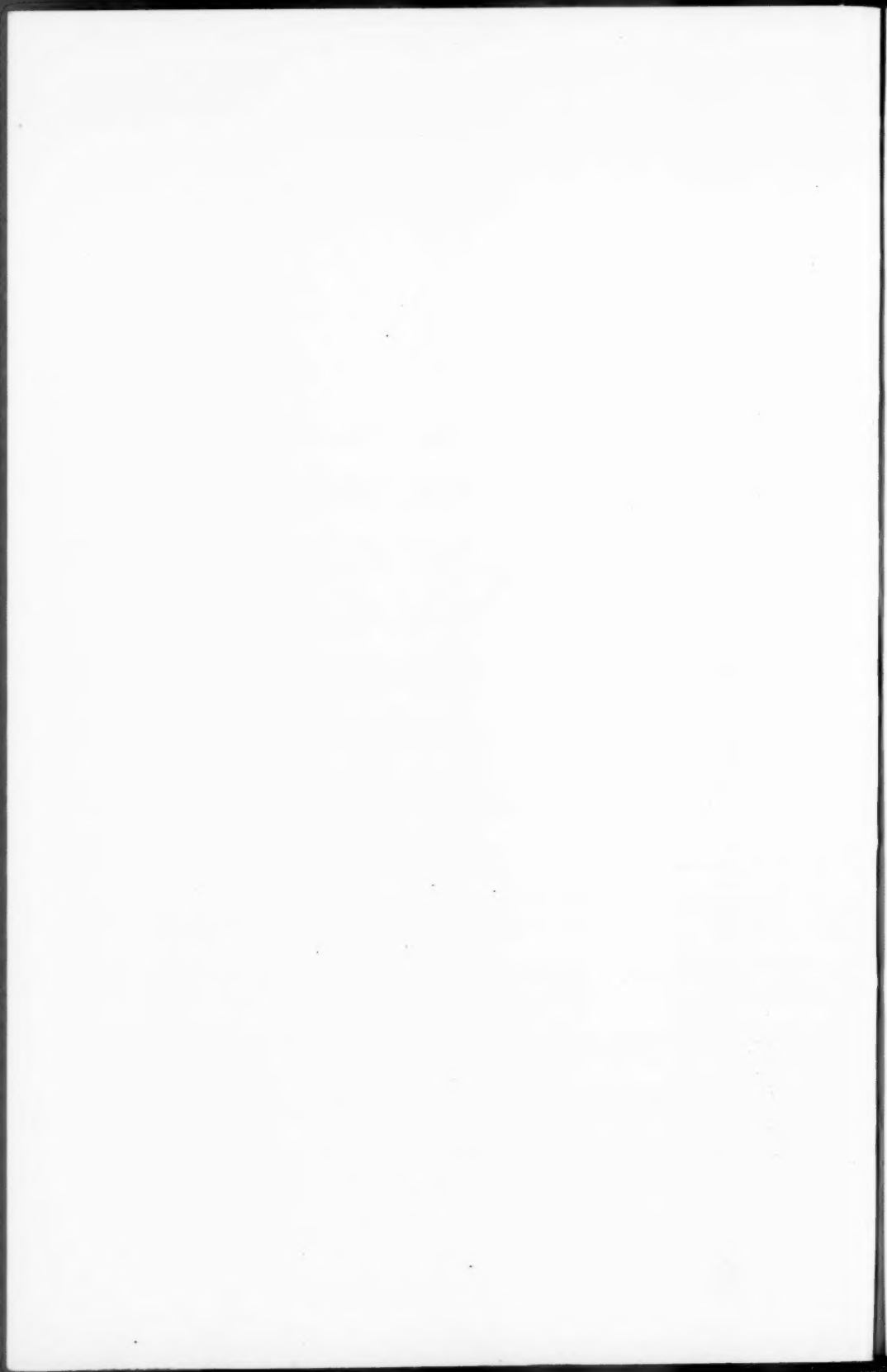
I have formed the opinion that subjects showing general lymphoid hyperplasia are more liable to die from trivial causes. I do not see at present how this is possible of proof, statistical or otherwise, as no one can determine the degree of triviality of the cause.

It is obvious that the diagnosis of status lymphaticus can be, and maybe is, abused and used to cover up a blunder or to conceal ignorance, but this applies to any form of dishonesty. I cannot think this happens often. The anæsthetist who turns on the carbon dioxide instead of the oxygen or the surgeon who cuts the aorta is not likely, I think, to be shielded by the pathologist who sees the picture of status lymphaticus with the eye of faith or friendship. I cannot feel, however, that any harm can be done if it is mentioned that a lymphatic condition is present, even if its exact meaning is not understood, in cases where there is no other obvious cause of death. If the terms "status thymico-lymphaticus" and "thymic death" were

abandoned and the simple term "lymphatism" used instead, there would be less misconception, and observations on these cases might be better directed and possibly lead to some solution of the problem which certainly does still exist.

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## Section of Odontology

President—F. St. J. STEADMAN, L.R.C.P., M.R.C.S., D.P.H., L.D.S.

[November 27, 1939]

### Antiseptics and Chemotherapy

By Professor ALEXANDER FLEMING, F.R.C.S.

THERE are very many odontological problems in connexion with antiseptics, as the mouth in many cases contains a considerable amount of tissue infected with various bacteria. During the last few years there has been no striking advance in "antiseptic" therapy, by which is meant the local application of an antibacterial chemical with a view to the direct destruction of the infecting bacteria. I will, therefore, limit my remarks on this aspect of the subject to a few general considerations which should always be borne in mind when using chemical antiseptics.

We must first accept that there are a great variety of chemical substances which can kill bacteria. That has been amply demonstrated, and the ordinary method of testing the potency of antiseptics—the Rideal-Walker test—is a comparison of their bactericidal action with that of carbolic acid on small numbers of microbes in a non-albuminous fluid. Such a test, while of value for some purposes, has no relation to the potency of antiseptics inside the body. The value of antiseptics outside the body is incontestable, and to take only one example, the chlorination of water has probably saved more lives than any other single hygienic measure. In the body, however, the case is entirely different, as here the antiseptic has to work under difficulties. In the mouth there are frequently masses of bacteria, and these bacteria are hidden away in pockets, and are surrounded by albuminous substances. We shall see later, in connexion with the sulphonamide group of chemicals, that the number of bacteria present makes an enormous difference to the result, and it is well known that most of the common antiseptics in use are greatly inhibited by the presence of albuminous substances. I have shown, also, the inability of the common antiseptics to sterilize a glass test tube with recesses much less complicated than are pockets around a tooth (Fleming, 1919).

The natural defences of the body have also to be considered, and it is of the greatest importance to assess the relative activity of chemical antiseptics on bacteria and on such natural defences. In the mouth there are two well-known antibacteria agencies—one, a ferment in the saliva, lysozyme, and the other, the polynuclear leucocytes and other phagocytic cells, which, as in other parts of the body, play a large part in resisting invasion.

The action of various chemical antiseptics on lysozyme has already been described (Fleming, 1931), but a very simple experiment may be cited as showing in a dramatic manner the complications of antiseptic therapy in the mouth. An ordinary culture plate of agar is planted with an organism sensitive to lysozyme action, and three holes are punched in the culture medium with a cork borer. Into one is placed a drop of saliva, into the second a drop of eusol, and into the third two drops of an equal mixture of saliva and eusol. After incubation a large zone of inhibition of growth is seen around the cavities containing the saliva and the eusol, but the mixture did not cause any inhibition of growth—the saliva and the eusol had "quenched" each other.

A test of the relative action of a chemical on bacteria and on leucocytes is, in my opinion, the best single test of the efficacy of an antiseptic for local or general use. The method has been described (Fleming, 1924, 1931), and it shows that with practically all the antiseptics in common use there is a concentration which destroys the leucocytes and allows the bacteria to grow out freely, and also that this anti-leucocytic concentration is very much less than that commonly used for treatment. This means that when these chemicals are used in the body in the usual concentrations all the phagocytic cells are destroyed before the bacteria are affected. Whenever it is found that a chemical destroys leucocytes more easily than it does a microbe there is little hope that it will have a beneficial action as a direct antiseptic in an infection by such microbe.

The older antiseptics had a greater affinity for leucocytes than they had for bacteria, but we have recently had at our disposal the sulphonamide group of chemicals. Sulphanilamide only affects leucocytic function in concentrations some twenty-five times greater than can be attained therapeutically in the human body, while it inhibits the growth of a sensitive microbe like *Streptococcus pyogenes* in concentrations only one-fiftieth of such therapeutic concentration. M & B 693 is likewise not injurious to leucocytes, and on *Streptococcus pyogenes* is something like ten times as potent as sulphanilamide. Such observations alone would make it practically certain that these chemicals would be effective in the treatment of *Streptococcus pyogenes* and, of course, this has been clinically established. In the investigation of new chemicals designed to combat bacterial infections in the body investigations of this kind should never be omitted, and if this were done we should have fewer extravagant claims and more truth in the advertisements of "antiseptic" chemicals.

This brings us to the subject of chemotherapy. It is now some thirty years since Ehrlich introduced salvarsan and this, and the allied arsenical compounds, revolutionized the treatment of spirochetal diseases, but the simpler bacteria proved more resistant. It has been the aim of every chemotherapeutist to produce a drug which would affect the ordinary bacteria which invade the body, and this has been in part achieved in the last few years by the sulphonamide compounds, especially sulphanilamide and M & B 693. These are now used most extensively in practice, and a large literature has grown up around them; but these drugs are so often misused that there are some practical points worth noticing.

(1) They are specific in their action; that is, they have a powerful action on some bacteria, but are without effect on others.

(2) When large numbers of even the most sensitive bacteria are present they have little or no antibacterial action.

(3) Peptone and bacterial extracts inhibit the bacteriostatic action of the sulphonamide compounds.

(4) The action is essentially bacteriostatic, and the natural defence mechanism of the body has to complete the destruction of the bacteria.

These points might be considered in more detail.

(1) *Specificity of the action of sulphanilamide.*—This can be demonstrated in many ways, but probably the simplest is by the growth of bacteria on ordinary culture plates in the presence of the drug. Streaks of various dilutions of a bacterial culture are made across a culture plate. Then a portion of the medium at right angles to the streaks is removed, and replaced by agar containing a strong solution of sulphanilamide. After incubation it will be seen that when the microbe (hæmolytic streptococcus) is sensitive to sulphanilamide there will be, when the implant of bacteria is reasonably small, a large area of complete inhibition of growth; but with an insensitive microbe, such as the enterococcus, there is no inhibition of growth, even with the smallest implant. This is brought out clearly in fig. 1.

The same thing can be done by mixing the chemical with infected human blood in slide cells, and noting the resultant growth. I have already (Fleming, 1939,

*Proceedings*, 32, 911, Sect. Obst., 71), illustrated what happens with sensitive and insensitive microbes in such an experiment. With the enterococcus there is growth even in a 1 : 200 dilution of sulphanilamide (about fifty times the concentration which is obtained therapeutically), while with the *Streptococcus pyogenes* (a sensitive microbe), inhibition of growth can be shown in a dilution of 1 : 500,000 (fifty times less than the therapeutic concentration). The difference, then, between a sensitive and an insensitive microbe may be enormous, and while it is reasonable to treat a patient suffering from an infection with *Streptococcus pyogenes* with sulphanilamide, it is

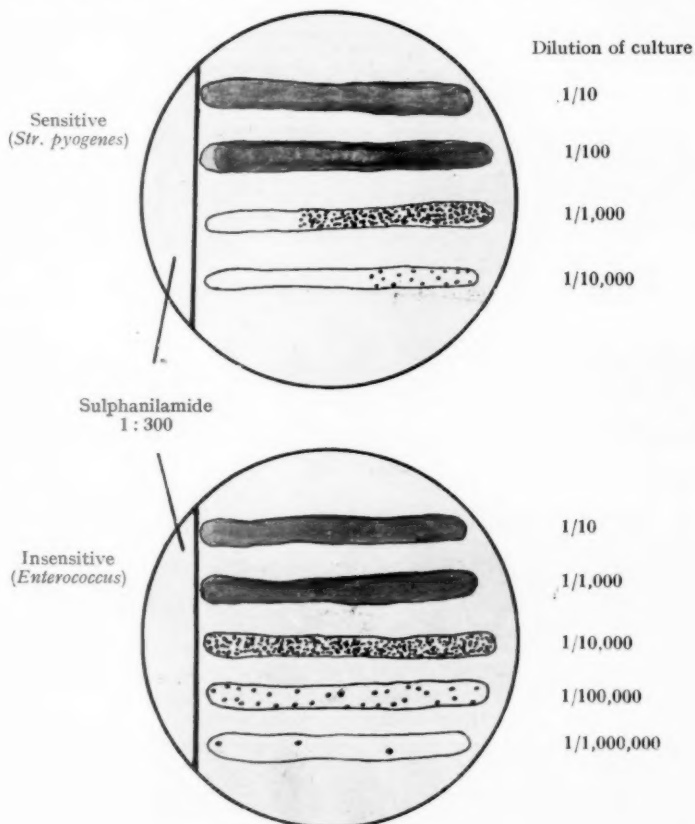


FIG. 1.—Specificity of sulphanilamide.

quite unjustifiable to embark on such treatment if the patient is infected with an insensitive bacterium, such as the enterococcus, as there would be no hope of doing good, but the usual risk of poisoning.

(2) When large numbers of bacteria are present sulphanilamide has little or no action.—This is shown on culture plates illustrated in fig. 1. Here, when the undiluted culture is streaked on the plate, it grows right up to the sulphanilamide, although, when it is diluted so that only a few streptococci are present, there is an area of inhibition of growth of several centimetres.

A more convincing experiment is that in which serial dilutions of sulphanilamide are mixed with human blood infected with various numbers of *Streptococcus pyogenes*,

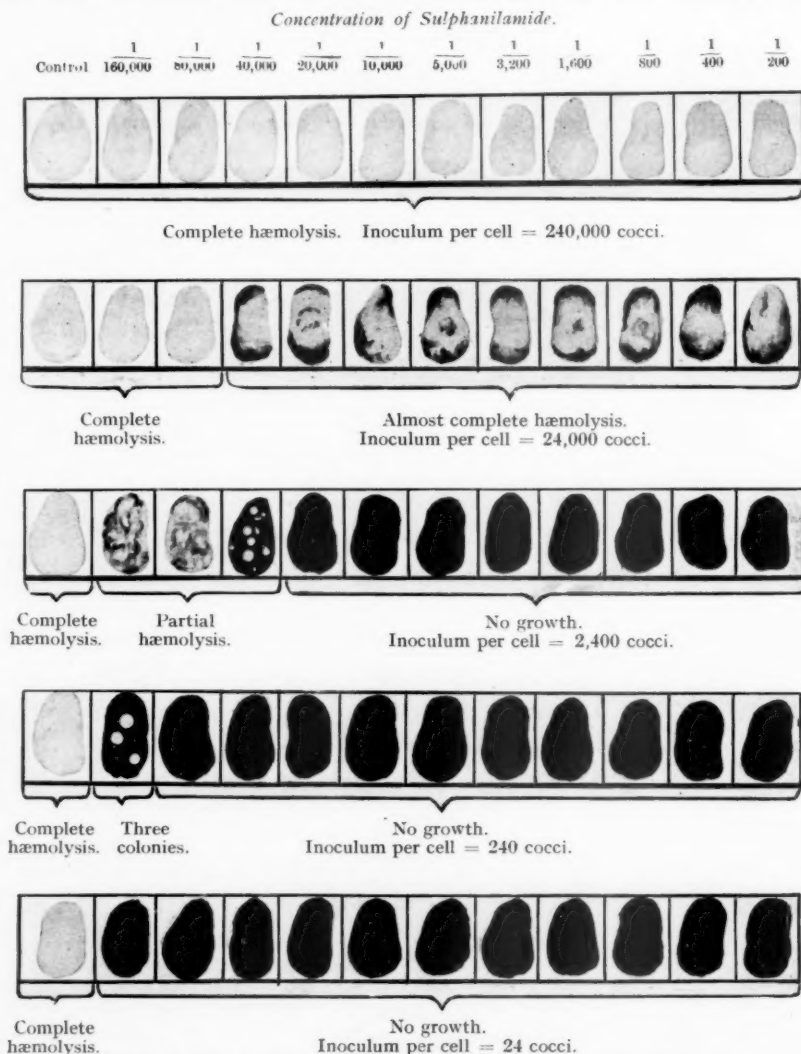


FIG. 2.—Antibacterial power of sulphanilamide on different numbers of hæmolytic streptococci.

and incubated in slide cells. The results obtained in such an experiment are shown in fig. 2.

Here it will be seen that, with the greatest inoculum of streptococci, growth and hæmolysis had taken place in eighteen hours in blood containing 1 : 200 sulphan-

ilamide (fifty times the therapeutic concentration), but as the numbers of streptococci got smaller the antibacterial action of the sulphanilamide was more and more marked, so that in the weakest streptococcal inoculum there was complete inhibition of growth up to a dilution of sulphanilamide of at least 1 : 160,000. It may be pointed out here that in this weakest streptococcal dilution, twenty-four cocci were planted into each cell, which contained 25 c.mm. of blood, so that the blood in each such cell contained 960 cocci per c.c., which is more than is usually found in the blood of a streptococcal septicæmia. This inability of the drug to act on large numbers of bacteria has been pointed out by Colebrook and others.

(3) *Bacteria and peptone inhibit the action of sulphanilamide.*—If large numbers of bacteria (dead or alive) are added to a saturated solution of sulphanilamide, and are then removed by centrifugalization, it is found that the supernatant fluid has no antibacterial action. It might be said that the chemical had entered into combination with the bacteria, and had been removed by centrifugalization, just as an agglutinin

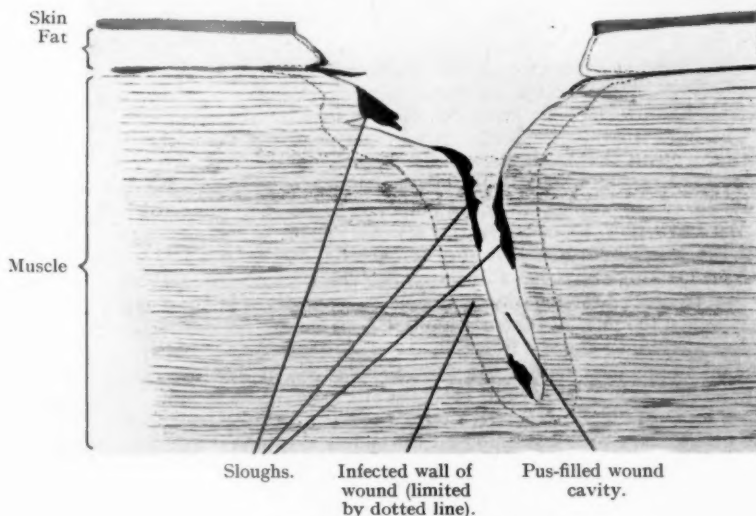


FIG. 3.—Diagram of a septic wound.

is absorbed by bacteria and comes down with them. This, however, is not the case, because if the supernatant fluid is diluted with normal salt solution the antibacterial power returns, and by further dilution it can be shown that none of the chemical has been absorbed by the bacteria.

Lockwood (1939) showed that when peptone, even in small quantities, was added to sulphanilamide, it quenched its antibacterial action, and I have, by experiments similar to those quoted above with bacterial suspensions, demonstrated that when this peptone sulphanilamide mixture is diluted with normal saline the antibacterial power returns, and that the peptone has merely inhibited the bacteriostatic action of the chemical. I have found that the peptone which I used exerted this action until the concentration was reduced to 1 : 5,000.

The practical importance of the presence of large numbers of bacteria and of peptone may now be discussed, and for this purpose we might take as an example a septic war wound. Fig. 3 represents a diagram of a war wound which has not been

excised, and which has become septic. Here we find a wound cavity filled with pus. Necrosed portions of muscle and connective tissue hang as sloughs from the walls of the wound, which are themselves infected to a considerable depth.

I have often been asked what would happen if sulphanilamide were applied as a dressing to a septic wound. Let us consider this point in the light of the experimental findings detailed above. The wound is filled with pus, which contains large numbers of microbes and pus cells. Some of these pus cells have broken down and liberated tryptic ferments, which act on the proteins present with the production of peptones. Therefore in the cavity of the wound there are masses of bacteria and peptones present, both of which completely inhibit the action of sulphanilamide, so that the conditions are the worst possible for the chemical to exert any antibacterial effect.

On these grounds it would be unreasonable to expect that the administration of sulphanilamide locally in a wound full of pus could influence the infection. Before we knew as much as we do about the action of the sulphonamide compounds I had an exceptional opportunity of observing the clinical effect of the local administration of sulphanilamide. A patient had an ulcer on each leg almost identical in size and position. These ulcers had persisted for several weeks, and they were both infected with a hæmolytic streptococcus which was sensitive to sulphanilamide. The surgeon in charge applied powdered sulphanilamide to one ulcer, and left the other alone. At the end of a week the ulcer which had not been treated with sulphanilamide had lost the hæmolytic streptococci and showed signs of healing, but in the one treated with sulphanilamide hæmolytic streptococci persisted, and healing had not commenced. The local treatment was abandoned, and sulphanilamide was given by the mouth, with the result that both ulcers healed rapidly.

It might, however, be argued that it would be quite easy to wash out the pus from the cavity of the wound, and that sulphanilamide then applied locally would succeed. We have now to consider the sloughs which cannot be washed away. These, like the pus, contain multitudes of bacteria and many broken-down cells which, in their breaking down, have liberated proteolytic ferments, and these in their turn have broken down proteins into peptones. The position in the sloughs is, then, exactly as it was in the pus, and there could be no reasonable hope of local application of sulphanilamide influencing the infection.

Sections of the infected wall of the wound show that in that portion of the wall nearest the wound cavity there are great numbers of bacteria and broken-down cells. Again, it could not be expected that if the wound cavity was filled with even a saturated solution of sulphanilamide that the chemical could influence the infection.

In view of these observations there does not seem to be a case for the local application of sulphanilamide to a septic wound.

Let us now consider what would happen in the septic wound when sulphanilamide is administered by the mouth or parenterally, so that it is absorbed and distributed throughout the body by the blood-stream. It would then attack first the outer portions of the infected wall of the wound, where there are the least numbers of bacteria and the greatest numbers of phagocytes. Here, then, the conditions are most favourable for the antibacterial action of sulphanilamide, and if the infection is by a sensitive microbe then it would be expected that the drug would affect the infection, as indeed it does. The few microbes present are inhibited by the sulphanilamide, and the multitude of phagocytes present rapidly deal with them. However, in the regions close to the cavity of the wound there are likely to be too many bacteria for the drug to have any effect, so one cannot expect a rapid sterilization of the wall of the wound. However, there will be a gradual eating away by the combined action of the chemical and the phagocytes, until the infection is conquered. It is unlikely that even prolonged administration of the drug will seriously influence the infection in the sloughs, or in the undrained cavity of a wound, even if the infection is by a sensitive microbe. The sloughs and pus must be dealt with surgically.

We have seen how sulphanilamide applied locally to a septic wound has little hope of success, and how the general application of the chemical is likely to prevent further invasion of sensitive bacteria, and gradually remove an infection with such sensitive bacteria from the walls of the wound, but not from masses of necrosed material.

At this stage mention might be made of a well-established procedure which seems likely to aid the action of sulphanilamide. It is the ordinary surgical practice to drain the cavity of a septic wound with a tube, or some similar contrivance. At least as important is it to drain the septic walls of a wound. Sir Almroth Wright long ago showed how this could be done with hypertonic saline, but little notice was taken of this until 1914, when in the Great War he republished his method and found many followers. The acutely inflamed walls of a septic wound contain much stagnant lymph which, by contact with bacteria, has lost the whole of its antibacterial properties. Hypertonic salt solution placed in the cavity attracts these stagnant fluids from the walls, and they are replaced by fresh fluids from the vessels containing their full complement of antibacterial substances.

I have published observations (1919) on the rate of transudation of fluid from a wound filled with hypertonic saline. A cup-shaped wound was chosen, into which a measured volume of fluid could be placed, and from which the whole of the fluid could be removed, measured, and examined at suitable intervals. First the normal rate of transudation was measured by emptying the cavity, and after a suitable interval, collecting the fluid which had collected in the cavity. Then when this had been removed, a measured volume of hypertonic salt solution was introduced. At intervals the amount of fluid in the cavity was measured, and the results obtained are shown in fig. 4. It will be seen that there is a very rapid rise in the rate of transudation of fluid into the wound cavity, and that this is gradually reduced as the strength of the salt solution is reduced.

In modern practice hypertonic saline solution has been to some extent replaced by magnesium sulphate paste, or glycerine, which have a similar "drawing" action on the walls of the wound.

The same result, but achieved by a different method, is obtained by a solution of hypochlorites, Dakin's fluid, or chloramine T. Observations were made as before in a cup-shaped wound, and the results are shown in fig. 4, alongside the result obtained with hypertonic saline. Here there is a latent period where the rate of transudation is not increased, and this is followed by a period of great increase. In this case the chemical causes a mild irritation of the walls of the wound which, after a short time, induces a greater outflow of fluid.

The reasons why this drainage of the walls of a wound would aid the action of the sulphonamide chemicals (sulphanilamide and M & B 693) are: It would "draw" from the infected walls of the wound stagnant fluids which contain large numbers of bacteria and products of protein disintegration, both of which inhibit the action of the sulphonamide compounds. These stagnant fluids have lost, also, their opsonic substances without which the phagocytic cells could not complete the destruction of the infecting bacteria even if their growth had been inhibited by the chemical. The place of these stagnant fluids is taken by fresh lymph from the vessels containing fewer bacteria, no protein breakdown compounds, and a full complement of opsonic substances—all tending to aid the action of the sulphonamide compounds. It seems obvious, therefore, that the drainage of the walls of a septic wound in the manner indicated must aid the action of, and should be used in conjunction with, treatment by the sulphonamide compounds.

Thus far we have discussed a septic war wound; fig. 5 is a diagram of a septic gum, which is essentially the same as the septic war wound. Here is a gum infected for some distance below the surface. There are pockets filled with pus containing, as does the pus of a septic wound, myriads of bacteria, and tryptic pus. The

sloughs of a wound are represented by tartar, and by food particles in which the bacteria grow and flourish. All I have said therefore about the septic wound applies equally well to the septic tooth.

*Sulphonamide therapy as an adjuvant to the surgical cleansing of wounds.*—There is another aspect of sulphonamide chemotherapy which applies especially to general surgery, but is of some importance in oral surgery.

In the past surgeons have been reluctant to operate on wounds infected with hæmolytic streptococci (*Streptococcus pyogenes*), as it was common experience that if fresh tissue planes were opened up to this most invasive organism there was a

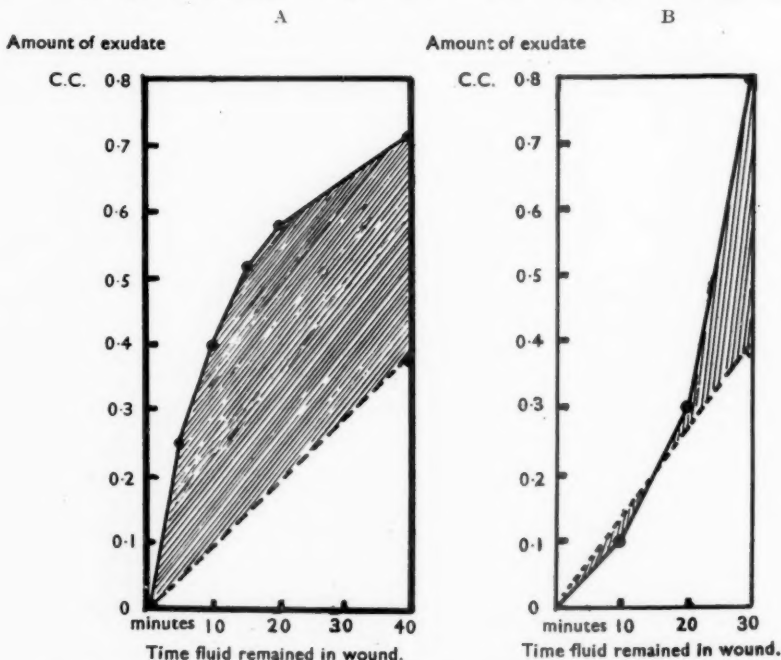


FIG. 4.—Rate of exudation of fluid into a wound cavity filled with (A) 5% salt solution; (B) 4% chloramine T. The broken line indicates the normal rate of exudation into the empty wound cavity. The continuous line shows the amount of exudate into the cavity filled with the chemical. The shaded area therefore gives the amount of exudate which is actually due to the application of the chemical.

serious risk of a generalized infection. In the Great War the infection of wounds with *Streptococcus pyogenes* was almost always a hospital infection (Fleming and Porteus, 1919) and there is no reason to suppose that in this respect things will now be different as no satisfactory method has yet been introduced for the prevention of spread of streptococcal infections in hospitals under war conditions.

The surgeons in this war have a great advantage over those in the Great War in that they have at their disposal chemicals like sulphanilamide and M & B 693 which can control hæmolytic streptococcal infections. This should make the surgery of septic wounds much simpler as, if sufficient of the chemical is administered to the patient so that his blood contains a concentration greater than that necessary to restrain the growth of small numbers of streptococci, it should be possible for the surgeon to operate on infected tissues without fear of spreading the streptococcal

infection. Thus the advent of the sulphonamide compounds has changed the whole outlook on streptococcal infections and should enable surgical wounds to be surgically cleansed with impunity. The importance of this will readily be realized by any surgeon who had to deal with *septic* wounds at a base hospital in the Great War.

(4) *The action of the sulphonamide drugs is essentially bacteriostatic, and the final destruction of the infecting bacteria has to be effected by the natural defensive mechanism of the body.*—I have demonstrated this with M & B 693 and pneumococci in the following experiment :—

Human blood was deprived of its leucocytes by filtering it through cotton-wool. By this procedure it completely lost its antipneumococcal power. This deleucocyted blood was then infected with pneumococci so that 25 c.mm. of blood contained 100

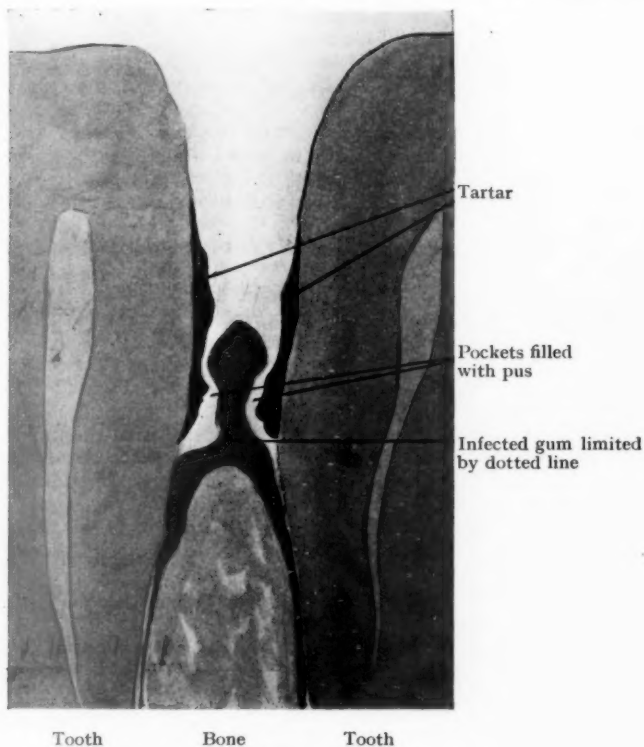


FIG. 5.—Diagram of periodontal infection.

pneumococci. Equal volumes of 25 c.mm. of this infected deleucocyted blood and serial dilutions of M & B 693 were mixed and incubated in slide cells. In all concentrations of the chemical up to 1 : 8,000, which is about the maximum which can be obtained therapeutically in the body, every pneumococcus grew out, so there was obviously no killing by the chemical, but the rate of growth was profoundly affected, as in any concentration greater than 1 : 128,000 the colonies were minute, and in the stronger concentrations they could not be detected by the naked eye, although with a low-power microscope they were clearly visible.

As the treatment of pneumococcal and streptococcal infections by M & B 693 is successful, something must kill the infecting bacteria, and if it is not the chemical

it must be the natural protective mechanism of the body. So far as the pyogenic cocci are concerned the body rids itself of infection by phagocytosis. For the completion of the work of the chemical, therefore, it is necessary that the phagocytic mechanism of the body should be efficient. The phagocytic cells should be active, and the body fluids rich in opsonin. If the phagocytic mechanism is seriously defective then, no matter how much of the drug is administered, the bacteria will not be killed, and although their active growth may be inhibited by the chemical, they will recommence to grow and produce their pathogenic effects when such administration is ceased.

It behoves us, then, to increase the immunity by every means in our power. This may be done by specific passive or active immunization methods, or by non-specific methods. Passive immunization is effected by antibacterial serums, active immunization by vaccines; and there are many non-specific methods of increasing the antibacterial power of the blood.

In dental practice there are few infections to which there is an effective antibacterial serum. In most cases, however, a specific vaccine can be prepared, and by the use of this a degree of immunity may be obtained which, in combination with the exhibition of a drug like sulphonamide or M & B 693, may make an enormous difference to the result. In this connexion I would like to cite an experiment which has been published (Maclean, Rogers, and Fleming, 1939).

Two rabbits were each inoculated with a single dose of 50 million pneumococcus vaccine. After six days these rabbits, together with two uninoculated rabbits, were infected with pneumococci. One of the inoculated and one of the uninoculated rabbits were treated orally with M & B 693, in doses of 1 grm. daily. Both of the rabbits not treated with M & B 693 died, and there was no evidence of increased immunity in the inoculated rabbit (it actually died sooner than the uninoculated animal). The uninoculated rabbit treated with M & B 693 became seriously ill with high temperature for four days and a positive blood culture, but eventually recovered. The inoculated rabbit treated with M & B 693 had only a trifling illness, and never had a positive blood culture.

This experiment indicates that a very slight degree of immunity can, in conjunction with the administration of M & B 693, make an enormous difference to the course of an infection. The vaccine by itself apparently did nothing, but in conjunction with M & B 693 it made such a difference that the vaccinated animal had only a trifling illness, while the uninoculated animal narrowly escaped death.

In practice, therefore, there are certain essentials which must be remembered in sulphonamide chemotherapy:—

- (1) If the infecting microbe is insensitive to the drug no good can result.
- (2) Local administration has little hope of success.
- (3) If infected dead material is present elimination of the infection is not to be expected.
- (4) Bacteria in abscesses are little likely to be directly affected by administration of the chemical.
- (5) Administration of the sulphonamide drugs should render safe the excision of a wound infected with *Streptococcus pyogenes*.
- (6) Methods are indicated which in septic wounds would prove valuable adjuvants to sulphonamide therapy:—

- (a) Drainage of the infected walls of the wound.
- (b) Increase of immunity, especially by vaccines.

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## Section of the History of Medicine

President—Sir WALTER LANGDON-BROWN, M.D.

### The Old English Medical Vocabulary

By CATHERINE LAMBERT

[This paper contains subject matter in Old English which made it impossible to be read aloud before the Section.]

OLD ENGLISH was by no means deficient in medical terms. Translations, or rather modified versions, of various Latin treatises on Medicine, such as the *Herbaria* bearing the names of Apuleius and of Dioscorides, and the *Medicina de Quadrupedibus* bearing the name of Sextus Placidus, fortunately have been preserved. In addition there are several 'leechbooks' in English of pre-Conquest date which are based on debased Latin compendia, which were themselves based on Greek originals. One of these leechbooks has been shown to be little more than a translation of a Salernitan original. Lastly there are a number of recipes and oddments found in different manuscripts or written as marginal notes or on fly leaves. These are our main sources for the Early English Medical Vocabulary, the outlines of which are here set forth.

The commonest words in Old English for illness, ill-health, were *untrumness* -*trymness*, and *mettrumness* -*trymness* :—

*Sodlice on ða ilcan wisan ðe oðer limo ðrowiað untrumnessa, se milte prowað on ða ilcan wisan*<sup>1</sup> ("Truly in the same way that the other limbs suffer ill-health the spleen suffers also").

The rare *untrymigo*, *untrumhad* and *untrymð* are also found with the same meaning.

*Gif hwylc wif seteð hire bearn ofer hrof oððe on ofen for hwylcere untrymðe hælo* (*alicujus morbi sanandi causa*<sup>2</sup>).

*Untrum* and *untrymig* meant ill, weak. *Ða wearð he untrum on feferadle*<sup>3</sup> ("Then he became ill with a fever-disease"). *Untrumian*, *untrymig(i)an* and *untrymman* are found for "to make or to become ill or weak". *Wearð he geuntrumod ond gewat to heofenan rice*<sup>4</sup> ("He became ill and went to Heaven"). The forms *mettrum* and *mettrumness* -*trymness*, are less common.

*Adl* was the most important word for disease in Old English, and its compounds were used for a number of different maladies. *Adl*, *morbus*.<sup>5</sup> There were more than twenty compounds of *adl*, *ceocadl* (cheek-disease), *feorhadl*, an interesting word meaning no specific malady, but simply "a mortal disease",<sup>6</sup> *fotadl* gout, *gealadl* jaundice, *lungenadl* lung disease, *maganadl* stomach disease, *wæteradl* dropsy. The adjective *adlig* is fairly common.

<sup>1</sup> Leon. 73/15. <sup>2</sup> L. Ecg. C. 33. Th. II. 156, 36. <sup>3</sup> Bl. Hom. 217/15. <sup>4</sup> Hom. Th. II. 348/4.

<sup>5</sup> Wrt. Voc. I. 45, 69. <sup>6</sup> Leon. 98/17.

Egrotas, adlige.<sup>7</sup> There was also a verb *adlian*,<sup>8</sup> the present participle of which *adligende*,<sup>9</sup> was used for 'a patient'.

An interesting word is Old English *coðu* which survived in literature till the fifteenth century, and still survives in dialect for a disease of cattle. There are several compounds which cover much the same ground as the compounds of *adl* but are fewer in number: *ban-* (bone), *bræc-* (epilepsy), *ear-*, *fof-* (foot), *heort-* (heart), *in-* (internal), and *muðcoðu* (mouth disease). The adjective *coðig* is a nonce-word used of sick sheep. *Coðige sceap* (oues moribide).<sup>10</sup> *Uncoðu* is a rather rare word for "disease, plague". *Us stalu ond cwalu, stric ond steorfa, orfwealm ond uncoða derede swyðe ðearle*<sup>10</sup> ("Theft and slaughter, plague and pestilence, murrain and diseases harmed us very severely").

Wulfstan's famous list of misfortunes includes more than one name for plague and pestilence, among them *stric* a rare word for plague (Latin *plaga*, stroke, blow). *Steorfa* was commoner and had the further meaning, "the flesh of animals that have died a natural death". *Færsteorfa*<sup>11</sup> is a rare word for "the murrain". The very rare *orfwealm* was also used for cattle disease. *Wol* was a very common word for "plague, pestilence". It has several derivatives. *Micel wol ond grim: acerba pestis*.<sup>12</sup> *Wolnes* appears once in the *Lorica*. *Wolnes fefor* (v.r. *woles*): *pestis febris*.<sup>13</sup> *Wolbærness* is a nonce-word.<sup>14</sup> *Wolberend* and *wolberendlic* are found for "pestilential, pestiferous", the latter being a nonce-word, the former not uncommon. *Se wolberenda* (*pestifer*) *stenc ðære lyfte monige dusende monna ond neata fordilgade & fornam*.<sup>15</sup> *Bewelan* is a rare word meaning to infect.<sup>16</sup>

Although *Wære* (pain), survives in dialect as *wark*, *warch*, there are several compounds that did not survive the Old English period. Such are *bladderwære*<sup>17</sup> (bladder-pain), *breost-*<sup>18</sup> (breast-), *fyllewære*<sup>19</sup> (epilepsy), *lendenwære*<sup>20</sup> (lumbago), &c. *Heafodwære*<sup>21</sup> survives in dialect as *headwark*<sup>21</sup> with a wide range of meaning. The derivatives of *wære* are nearly as numerous as those of *adl* and amount to much the same thing.

*Lef*, *gelefed*, are found for "injured, infirm". *Lef, debilis*.<sup>22</sup> *Swa bið eac on wintra for cyle ond for ðara wedra missenlicnesse ðæt se milte wyrð gelefed*<sup>23</sup> ("So it is also in the Winter, on account of the cold and the changes in the weather that the spleen becomes weakened [or injured]"). This word is also found signifying aged, that is weakened by age.<sup>24</sup> *Lefung*<sup>25</sup> is a nonce-word for weakness, paralysis.

*Misbyrdo* is a very rare word signifying the imperfection of a bodily organ. *Læcedomas* *be wambe missenlicre gecyndo oððe ðære misbyrdo* ("Remedies for various forms of birth, or for ? imperfections of the womb, or ? abortions"<sup>24a</sup>). Other bodily imperfections appear as *hoforode* and *healede*. *Gef (wif) eteð fearres flæsc oððe rammes... ðonne gelimpeð hit hwilum ðurh ðæt cild bið hoforode ond healede*<sup>29</sup> ("If a woman eats the flesh of swine or rams, then it sometimes happens through this that the child is hump-backed and ruptured").

*Hærseard* is a nonce-word for "hare-lip". *Læcedom wið Hærserde*.<sup>26</sup>

*Sceoleagede*, *-ige*, signifies cross-eyed, squinting, glossing *strabus*,<sup>27</sup>

<sup>7</sup> AN. OX. 1977. <sup>8</sup> Lchdm. III. 151. <sup>9</sup> Ib. I. 184. <sup>10</sup> Nap. Contr. 277. <sup>11</sup> Lchdm. III. 56/15, 25. <sup>12</sup> Bd. I. 14. Sch. 39, 806. <sup>13</sup> Leon. 192. XXXIII. <sup>14</sup> Oros. 2, 1: Swt. 62. 34. <sup>15</sup> Bd. I. 13. Sch. 37, 765. <sup>16</sup> Ps. Vos. 105, 38. <sup>17</sup> Leon. 91/31. <sup>18</sup> Ib. 96/40. <sup>19</sup> Ib. 56/6. <sup>20</sup> Ib. 72/3. <sup>21</sup> Wrt. D.D. "Head." <sup>22</sup> Germ. 389. <sup>23</sup> Leon. 73/27. <sup>24</sup> Bd. 3, 8. Sch. 222, 754. <sup>24a</sup> Lchdm. II. 220. <sup>25</sup> Hom. Th. II. 486/18. <sup>26</sup> Leon. 2/8. v. 18/8. <sup>27</sup> Wrt. Voc. I. 45, 56.

torbus<sup>28</sup> and scevus.<sup>29</sup> Siwenige, -ege and tor(e)nige are two obsolete terms signifying blear-eyed. Siweneg:lippos.<sup>30</sup> Gif he wære torenige oððe fleah hæfde on eagan: si lippos fuerit, si albuginem habens in oculo.<sup>31</sup>

Fleah, fleo, flie, is found several times for a white spot in the eye.<sup>32</sup>

Æsmæl is a rare word for "a contraction of the pupil of the eye". The following quotation gives æsmæl among other eye troubles: Læcedomas wið eallum tiedernessum eagna, wið eagna miste . . . ond wið flie ond wið eagna tearum ond wið wemme on eagum, ond wið æsmælum, ond gif mon surege sie, wið pocces on eagum ond wið gefigom ond wið wýrmum on eagum, ond eagsealfa ælces cynnes<sup>33</sup> ("Remedies for all weaknesses of the eyes, for mist [for a cloud] in the eyes, and for a white spot, and for tears in the eyes, and for a blemish on the eyes, and for contraction of the pupil, and if one is blear-eyed, for pustules in the eyes, and eye-salves of every kind").

Unscearpsiene is a very uncommon word for weak sight. Ealdes mannes eagan beoð unscearpsyne . . . ðus mon sceal unscearpsynum sealf wyrcean to eagum<sup>34</sup> ("An old man's eyes are weak-sighted . . . one should thus make an ointment for the eyes of the weak sighted").

Although the word gund survived as late as the seventeenth century, meaning foul matter secreted in the eye, the compound healsgund meaning a swelling in the neck, is not found after the Old English period. Ad parotidas ðæl ys to ðan sare ðe abutan sa earan wýest ðat man nemned on ure geðeode healsgund<sup>35</sup> (" . . . that is for the sore that develops about the ears that is called swelling in the neck in our language").

There were many names of diseases and symptoms in Old English apart from those already referred to as compounds of adl, codu and wære. Of these perhaps the most interesting are the old words for fever, since among them it is possible to watch a native word becoming obsolete in the Old English period. A cursory examination of the Old English medical literature will show how very much commoner was the Latin loan-word fefer than any native term. Not only was fefer used in combination with native words to form compounds such as feferadl<sup>36</sup> (fever disease), fefercynn<sup>37</sup> (kind of fever), but a verb, feferian<sup>38</sup> (to be feverish) was formed.

Rare Old English words for fever were drif, gedrif, hrið, hruð, hriðing, hriðadl, bryneadl and lenctenadl. Drif is found once at the end of the Old English period; gedrif is slightly commoner. Full-neah æfre ðe oðer man wearð on ðam wyrrestan yfele ðæt is on ðam drife<sup>39</sup> ("Very nearly every other man was in the worst trouble, that is the fever"). Fefer . . . ðæt is micel hæto ond hruð<sup>40</sup> ("Fever . . . that is a great heat and fever"). Gif him hriðadl getenge bið, ða todrifð se wyrtdrence<sup>41</sup> ("If fever occurs the herbal draught will drive it away"). Hriðing<sup>42</sup> is a nonce-word, and the verb hriðian<sup>43</sup> is very rare.

There are various forms of fever. Febris a fervore nominatur id est bryneadl.<sup>44</sup> Lenctenadl, is found for special types of fever, and once for dysentery. Lengtenadl, tipus,<sup>45</sup> Lenctenadl, tertiana,<sup>46</sup> Hara bið god wið lengtenadl (contra dysenteriam)<sup>47</sup> ("Hare is good for dysentery").

<sup>28</sup> Txs. 98, 981. v. 99, 1939. <sup>29</sup> Lchdm. III. 144. <sup>30</sup> Germ. 396, 284. <sup>31</sup> Past. II. Swt. 65, 5. <sup>32</sup> Leon. 11/5, 9, 10, &c. <sup>33</sup> Ib. 1/5. v. 12/20. <sup>34</sup> Ib. 19/33-38. <sup>35</sup> Lchdm. III. 94. v. Leon. 14/26. <sup>36</sup> Bl. Hom. 217, 16: 227, 5. <sup>37</sup> Leon. 5/11. <sup>38</sup> Lchdm. I. 122, 220. <sup>39</sup> Chr. P. 1086, p. 217. <sup>40</sup> Leon. 64/20. <sup>41</sup> Ib. 64/28. <sup>42</sup> Ib. 77/23. <sup>43</sup> Ib. 66/8-10. <sup>44</sup> Wrt. Voc. II. 39/9. <sup>45</sup> Wrt. Voc. 19, 30. <sup>46</sup> Ib. 289, 58. <sup>47</sup> LL. Th. II. 162, 23.

Dysentery appears also as *utsiht*,<sup>48</sup> *utsihtadl*, and *utwærc*. Thus:—  
*Sio utsihtadl cymð manegum of to miclum utgange,*  
*ond ðonne lange hwile ne gymð mon ðæs oððæt se innod*  
*wyrð ge onburnen ge ðurh ðæt gewundod*<sup>49</sup> ("Dysentery comes  
 to many first from too violent actions of the bowels; then for a long time they pay  
 no attention to this until the intestines are inflamed and are injured by that").

The rare *Metetsiht*<sup>50</sup> is found for a disease which causes the food to pass  
 through the bowels undigested.

Obsolete terms for "inflammation, pus, and purulent", are represented in Old  
 English by *lyswn*,<sup>51</sup> as noun and adjective, *om*, *omig*, *omiht*, and  
*dylsta*, *dylstiht*. *Ðonne se swile tobyrst ðonne bið seo*  
*mige lyswn swilce worms*<sup>52</sup> ("When the swelling bursts then the  
 urine is purulent like corrupt matter"). The plural form *oman*, derived from  
*om*, *rust*, is found signifying an erysipelatous condition. *Igni-sacrum*,  
*oman*.<sup>53</sup> The compound *healsome* is found once. *Se man se ðe bið*  
*on healsoman nime healswyrð*<sup>54</sup> ("The man who has inflammation  
 of the neck should take *Campanula Trachelium*"). *Omig*, literally rusty, and  
*omiht*, seem to signify inflammatory. *Gif ðæt sie omihte wæte innan*  
*onburnenu tyhte hie mon ut mid liðum mettum sincendum*  
*ond ne læt inne gesittan on ðam lichoman ond wyrð*  
*gegaderodu omig wæte on ðære wambe*<sup>55</sup> ("If there be  
 inflammatory moisture within, it should be drawn out with food that acts as a  
 gentle aperient, and not be left in the body [to cause] inflammatory moisture to  
 collect in the stomach"). *Omcyn* occurs once for pus.<sup>56</sup> *Dylsta*<sup>57</sup> and  
*dylstiht*<sup>58</sup> are very rare.

*Ðeor* is the name of an unidentified diseased state. Cockayne translates  
 'dry disease', which is not illuminating.<sup>59</sup> The *Læceboc* provides many prescriptions  
 for the 'theor', among them a *Sealf wið ðeore*<sup>62</sup> ("Ointment for theor").  
 A draught is also prescribed for the 'theor' and for a shooting wen, or tumour', and  
 a fomentation 'if the theor remains in one place'.<sup>60</sup> *Ðeorgeryde*, implying  
 the inflammation caused by theor, is found once. *Arestolobius... gesette*  
*... godne morgendrænc... se drænc is god wið oðrum*  
*giccendum blece ond ðeorgeride*<sup>61</sup> ("Aristobulus appointed a good  
 morning-draught... the draught is good for other irritating skin diseases and the  
 theor inflammation"). *Ðeoradl* is also found but unfortunately explained by  
 another equally obscure word, *gefigo*. *Wio ðeoradl on eagum ðe*  
*mon gefigo hæton læden hatte cimosi*<sup>63</sup> ("For theor-disease  
 in the eyes, that is called *gefigo*, in Latin it is called *cmosis*"). *Cimosi* here stands  
 for chemosis. A hot compress made from the red nettle is prescribed for 'theorworm  
 in the feet',<sup>64</sup> and a poultice for a *ðeorwenn* (theor-tumour) on the knee.<sup>65</sup>  
*Ðeordrenc* appears once for a theor-draught.<sup>66</sup>

Symptoms being described either inadequately or not at all, it is often impossible  
 even to guess at the real nature of the condition from the treatment prescribed, there  
 being often great similarity in the remedies proposed for different diseased states.  
 Deductions may sometimes be drawn when two diseased states are coupled but  
 it is not safe to push this method very far. Occasionally the Greek or Latin  
 name descriptive of the condition is mentioned. Thus *Titanus*, *ofer-*  
*bæcgeteung*<sup>67</sup> and there is a recognizable description of tetanus in the medical  
 literature.<sup>68</sup> There were, in many cases, a number of slightly different names for the

<sup>48</sup> Leon. 83/25. <sup>49</sup> Ib. 83/34. <sup>50</sup> Wrt. Voc. I. 19, 54. <sup>51</sup> Leon. 15/12. <sup>52</sup> Ib. 60/8. <sup>53</sup> W.W. 26/4; 421/41. <sup>54</sup> Lchdm. II. 218. <sup>55</sup> Leon. 65/34-37. <sup>56</sup> Ib. 26/3. <sup>57</sup> Leon. 22/49. <sup>58</sup> Ib. 22/12. <sup>59</sup> Payne 48. <sup>60</sup> Leon. 92/9. <sup>61</sup> Leon. 150/19-151/1. <sup>62</sup> Leon. 36/24. <sup>63</sup> Leon. 12/30. <sup>64</sup> Ib. 36/36. <sup>65</sup> Ib. 104/38. <sup>66</sup> Ib. 99/19. <sup>67</sup> W.W. 112/20. <sup>68</sup> Lchdm. III. 110.

same condition. Thus dropsy appears as wæteradl, wæterseocness, and wæterbolla.<sup>69</sup>

Onflyge, the "on-flying things" was a word for an infectious disease. It occurs three times in a charm against diseases, pains, poisons, and 'worms'. This charm is a valuable relic of the old Teutonic medicine and magic, as distinguished from the classical traditions or documents, for it is the latter that lie at the back of most of the Old English medicine that has survived. Ðu miht wið attre ond wið onflyge<sup>70</sup> ("Thou hast power against poison and against infectious disease"). Geflog is also found with the same meaning in this passage, the old idea being that infection was blown on to a person from the air.

Internal diseases appear as innoðsar,<sup>71</sup> -tiederness,<sup>72</sup> -wund,<sup>73</sup> inadl, and incoðu. There is little or no possibility of distinguishing between these diseases, except that inadl is sometimes used for internal disease in general<sup>74</sup> and once specially for diseases of women,<sup>75</sup> and incoðu<sup>76</sup> is found glossing melancholia, and once possibly for "fever", though the ambiguous Latin is *fibras*.<sup>77</sup> Incoða is also found for *incommoditates*, *inconvenientias*, *l infirmitates*<sup>78</sup> while Incund,<sup>79</sup> inne-, innancund<sup>80</sup> are found simply for 'internal.'

There are numerous obsolete words for mental disease in Old English. Such are monaðseocness, wedenheortness, ungewitfæstness, ungemynd, modeselhygd, and many compounds of wod which itself survives in dialect as wood. Lunacy is represented by monaðseocness, wedenheort, -nes.

Wið monaðseocnysse gyf man ðas wyrte peoniam ðam monaðseocan ligcendon ofer alegð sona he hyne sylfne halne upahefð.<sup>81</sup> ("For lunacy, give the herb peony to the lunatic when he is recumbent and lay it upon him; he will at once raise himself, cured").

Hi ongunnon ðæt hi his wedenheortnysse gestilldon: motus ejus insanos comprimere conati.<sup>82</sup>

Wedenheort meant both "madness" and "mad". Leoht drenc wið wedenheorte ("A mild draught for madness"). Weden(d)seoc is also occasionally found<sup>83</sup> Wodðraga, vesania.<sup>84</sup> Widdendream and wedenonfa are curious survivals in Scottish dialect of Old English wodenwoddream and there are some later combinations of weden and onfall.

From a list of the symptoms of the *sidansar* that is 'disease in the side,' comes modeselhygd<sup>85</sup>; this must mean delirium. Brægenes hwyrftness may mean either delirium or lunacy. 'A good morning-draught' is prescribed for this.<sup>86</sup>

Gewitleast, "folly, madness". Wið ða adle ðe grecas frenesis nemnað ðæt is on ure geoeode gewitleast ðæs modes ðæt byð ðonne ðæt heafod aweallan byð<sup>87</sup> ("For the disease which the Greeks call frenesis, that is, in our language, madness of the mind, that is when the head is on fire").

Gedwolding is a rare word apparently for delusions; ðis is balzaman smyring wið eallum untrumnessum ðe on mannes lichoman bið wið fefre ond wið seinclace ond wið eallum gedwoldinge<sup>88</sup> ("This is a balm ointment for all the illnesses of man's body, for fever, and for ghosts, and for all delusions").

Bræcseoc meant either epileptic or lunatic. Gif mon sy ðære healf-

<sup>69</sup> Leon. 50/1, 27, &c. <sup>70</sup> Lchdm. III. 32. v. III. 36. <sup>71</sup> Leon. 4/22. <sup>72</sup> Ib. 52/32. <sup>73</sup> Ib. 49/39. <sup>74</sup> Leon. 50/33. <sup>75</sup> Ib. 53/8. <sup>76</sup> Hpt. Gl. 478. <sup>77</sup> Ib. 453. <sup>78</sup> Ib. 453. <sup>79</sup> Lchdm. I. 196. <sup>80</sup> Leon. 140/27-30. <sup>81</sup> Lchdm. I. 170. <sup>82</sup> Bd. 3, 11: Sch. 140, 1189. <sup>83</sup> Gr. D. 135, 1, 223, 22. <sup>84</sup> Past. 26; Swt. 183/24. <sup>85</sup> Leon. 77/37. <sup>86</sup> Ib. 150/20. <sup>87</sup> Lchdm. I. 208-210. <sup>88</sup> Leon. 87/13.

deadan adle seoc oððe bræcseoc<sup>89</sup> ("If one be sick of the 'half-dead' disease, or epileptic"). Bræccōðu, Epilepsia uel caduca, uel laruatio, uel comitalis, bræccōðu, fylleseoc.<sup>90</sup>

Modes geswæðrung<sup>91</sup> appears for "loss of mental powers".

With the exception of blood-letting there are small traces of surgery in the Old English medical literature. Instructions for an amputation are given in the *Læceboc*, but no obsolete words are used for the operation.

Ofcyrf is found once in the Homilies for "an amputation". Hwæt getacnað ðæs fylmenes ofcyrf?<sup>92</sup> ("What does the amputation of the membrane signify?"). Snide<sup>93</sup> is found for "an incision", and snidisen for "a lancet". Hrin ðu him mid ðy snid isene ond snið lythwon ond listum ðæt ðæt blod mæge ut furðum<sup>94</sup> ("Touch him with the lancet and cut slightly and skilfully so that the blood may also run out").

Dolgs waðu<sup>95</sup> and ora are found for a scar, and dolgdrenc<sup>96</sup> is a potion for a scar. Dolgdrenc is also found once signifying antidote.<sup>97</sup>

Bloddolg means the wound made by the operation of blood-letting. There are instructions for procedure "if the wound made by blood-letting turn bad".<sup>98</sup>

Blood-letting, which was frequent in Old English medical treatment, appears as blodlæs.<sup>99</sup> Blodlæstid is "the season for blood-letting".<sup>100</sup>

There are several obsolete words for hæmorrhage, blodsihte,<sup>101</sup> blodgyte,<sup>102</sup> and blodryne.<sup>103</sup> The last is once found for apoplexy. Gefor he on blodryne: effusione sanguinis, quod Græce apoplexis vocatur mortuus est.<sup>104</sup> Blodseten<sup>105</sup> signifies a remedy to stop bleeding. Blodseax, æderseax, were names for a "lancet", they gloss flebotomum.<sup>106</sup>

There were a few words for "fracture". Banbryce<sup>107</sup> is found for fracture of a bone. A herb salve with honey is prescribed for a fractured bone in the head.<sup>107</sup> Forod<sup>108</sup> also signifies fractured, and a herbal ointment, mixed with white of egg, is prescribed for a broken leg.<sup>108</sup> Scane forod (leg-fractured), and forod fot<sup>109</sup> (fractured-foot) are nonce-words.

Dislocation seems implied by the phrase, Gif sio eaxl upstige<sup>110</sup> ("If the shoulder rise up"). The remedy proposed is an ointment.

Sepsis can have been no uncommon accompaniment of pre-Conquest surgery, and is frequently indicated. There was a word for it, unsyfre, and it is used in connexion with the treatment of wounds caused by blood-letting. Donne sio wund sie clæne geryme ðonne ðæt ðæt ðyrel to nearo ne sie . . . gif hio swiðor unsyfre weorpe (sic) clæsna mid hunige ond gelæteft togædre<sup>112</sup> ("When the wound is clean, enlarge it so that the aperture be not too narrow . . . if it becomes very septic, cleanse it with honey and close it again"). Unsyferness means impurity, physical or moral. It is found for "the impurities of the blood" in "disease of the liver".<sup>111</sup>

There were a number of minor ailments for which Old English medicine provided remedies. Chilblains, cramp, insomnia, and singing in the ears, are the chief of these. A herb salve compounded with honey is at least harmless for chilblains, but the remedy for insomnia is distinctly more adventurous. To slæpe wulfes heafod lege under ðone pyle, se unhæla slæpeð<sup>113</sup> ("In order to sleep, put a wolf's head under the pillow, the invalid will sleep").

There is no Old English term covered by our word diagnosis, but a few words can be found describing the processes which lead up to the doctor's verdict. Thus

<sup>89</sup> Ib. 86/8. <sup>90</sup> W.W. 112/26. <sup>91</sup> Leon. 62/7. <sup>92</sup> Hom. Th. I. 94/32. <sup>93</sup> Leon. 5/34. <sup>94</sup> Ib. 62/36. <sup>95</sup> W.W. 335/10. <sup>96</sup> Leon. 100/8. <sup>97</sup> Hpt. Gl. 15. <sup>98</sup> Leon. 5/34. <sup>99</sup> Ib. 5/32. <sup>100</sup> Ib. 44/35. <sup>101</sup> Leon. 52/16. <sup>102</sup> Lchdm. I. 88. <sup>103</sup> Ib. 180. <sup>104</sup> Oros. 6/33; Swt. 288/27. <sup>105</sup> Leon. 17/26. <sup>106</sup> Wrt. Voc. II. 39/22. <sup>107</sup> Leon. 28/38. <sup>108</sup> Leon. 21/16. <sup>109</sup> Past. XI. Swt. 66/12. <sup>110</sup> Leon. 100/7. <sup>111</sup> Ib. 52/29. <sup>112</sup> Ib. 63/3. <sup>113</sup> Lchdm. I. 360.

Orðanc is found once for experiment or test. Gyf ðu ðonne mid orðance ðissēs ðinge fundian wille, gecnuca ða wyrte ond wrið hy to ðinne halan handa, sona heo yt ðone lichaman<sup>114</sup> ("If you would prove this by experiment, pound up the herbs and bind them to your sound hand: soon it will eat into your body"). Orfundelness is a very rare word for experience or proof. ðeh ðu hyre leaf ond hyre wyrtruman do on anne clænne clað ond gewriðe onbutan ðæs mannes swyran ðe ðæt yfel ðolað, hyt deð onfundelnyssse ðæs sylfan ðinges<sup>115</sup> ("If you put the leaf and the roots in a clean cloth and bind it round the neck of the man who is suffering from the trouble, it will give you a proof of the same thing").

Beotung, a threatening, is found once, in the plural, for threatening symptoms. Be wambe coðum . . . ærest hire bið on innan wund, ðonne bið ðær sar ond beotunga ond gesceorf<sup>116</sup> ("For diseases of the stomach . . . firstly, if the stomach is injured internally, then there are soreness there and threatening symptoms and a gnawing").

Ungewendendlic<sup>117</sup> and singal are each found once for "chronic". Wið ealda ond singalum heafodece<sup>118</sup> ("For old and chronic headaches").

Eaðlæce, -læcne<sup>119</sup> meant easy of cure; uneaðlæce,<sup>120</sup> -læcne, the contrary. Unlæcnigendlic appears once for incurable. ðeos wyrte soðlice ealle ealde ond hefige ond unlæcnigendlice adlu tofered swa ðæt he byð gelæcnud ðeah he ær his hæle on tolætennesse wære<sup>121</sup> ("This herb truly overcomes all old-established and severe and incurable diseases, so that he is cured though he had formerly despaired of cure"). The meaning of læcnigendlic is, not so much curable, as susceptible of medical treatment. It occurs only once. Mid læcnigendlicum tolum, instrumentis medicinalibus.<sup>122</sup>

Although the word leech is still extant, as equivalent to physician, a number of compounds that are obsolete are found, læcegetawu<sup>123</sup> (medical instruments); læceiren<sup>124</sup> (medical iron instrument); læceseax<sup>125</sup> (lancet); læceist<sup>123</sup> (medicine chest). Læcewyrte, the name of a plant, is also found as a general term for medical treatment. Min adlige cneow is yfele gehæfd, ðæt ne mihte nan læcewyrte awiht geliðian, ðeah ðe heo gelome togeled wære<sup>126</sup> ("My diseased knee is in bad condition, so that no medical treatment can soothe it at all, although it were often applied").

Anspilde appears once as equivalent to salutary. ðæt bið anspilde lyb wið eagna dimnesse<sup>127</sup> ("That is a salutary medicine for dimness of the eyes").

Fultum, a very common word for help, assistance, is used specifically for a medical remedy. Gif ðas fultumas ne syn helpe, læt blod ðonne on ædre of earme<sup>128</sup> ("If these remedies are of no use, then let blood from the vein of the arm").

The actual remedies in the texts are innumerable, external and internal, animal, vegetable, and mineral. We turn to consider some of the more general terms for them.

Lybb, which meant both poison and magic in Old English, is used also for medicine. See quotation above, for 'dimness of the eyes'.<sup>127</sup> But its opposite unlybba is used for poison in this text and never lybb. Wið unlybbum supecubutan<sup>128</sup> ("For poisons drink the butter of cows"). The compound

<sup>114</sup> Lchdm. I. 100. <sup>115</sup> Ib. I. 160. v. 140. <sup>116</sup> Leon. 66/6. <sup>117</sup> Lchdm. 328. <sup>118</sup> Ib. I. 380.

<sup>119</sup> Leon. 86/1. v. 86/6. <sup>120</sup> Ib. 78/7. <sup>121</sup> Lchdm. I. 262. <sup>122</sup> Hpt. Gl. 478. <sup>123</sup> Gr. D. 344/16.

<sup>124</sup> Ib. 32/25. <sup>125</sup> Past. 26/3; Swt. 187/9. <sup>126</sup> Hom. Th. II. 134/33. <sup>127</sup> Leon. 10/22. <sup>128</sup> Ib. 89/4.

lybceorn describes "a grain of purgative effect, especially the seed of various euphorbias and probably also the seeds of some of the gourds". Libbceorn catharticum.<sup>129</sup> Drenc, extant as drench, a potion given to animals, gave rise to a good many compounds. Such are, slæpdrenc<sup>130</sup> (sleeping-draught); clænsungdrenc<sup>131</sup> (cleansing draught), eceddrenc<sup>132</sup> (vinegar draught); spiw.<sup>133</sup> and wece-drenc<sup>134</sup> for an emetic. Spiwol is also found for emetic,<sup>135</sup> its contrary, unspiwol,<sup>136</sup> is rare.

Vinegar was often prescribed in Old English medicine. The obsolete word is eced. Wið innodes hefignessesyle etan rædic mid sealte ond eced supan, sona bið ðæt mod leoh tre<sup>137</sup> ("For heaviness of the stomach, give radish with salt, and vinegar to drink, the mind will be easier at once").

Posel,<sup>138</sup> posling,<sup>139</sup> and clyne,<sup>140</sup> are found once each for "pill".

Fasting, which is often recommended in these prescriptions, appears as nihtig, nihtstnig. Wið angebreoste wyll holerinde on gate meolce ond sup wearne nyhstig<sup>141</sup> ("For asthma, boil the rind of holly [or alder tree] in goat's milk and drink warm, when fasting"). The compound nihtnihtig is also not uncommon. Selle mon neahtnestigum<sup>142</sup> ("Give it to a man fasting at night").

Smereness is the ordinary word for "an ointment". Læcedomas wið canceradl, ðæt is bite, ond smerenessa ond sealf<sup>143</sup> ("Treatments for the cancer disease, that is biting disease, and an ointment and salve"). Ðwænan signifies to soften by moisture or by the application of an ointment. Rysele oððe gelynde wið garleac gemenged ond on aled ðone swyle ðwænð<sup>144</sup> ("Fat or grease mixed with garlic and laid on the swelling softens it").

There are several rare words for a bandage; sarclað, seaxclað, wætla (cognate with modern English wattle), and wræd, a poetical word for "band, fillet". Sarclað, fasciola.<sup>145</sup> Seaxclað oððe wræd wriðels, fascia.<sup>146</sup> Hafa ðe linenne wætlangearone ðæt ðu ðæt dolh sona mid forwriðe<sup>147</sup> ("Have the linen bandage ready so that you bind up the wound with it at once"). Gewriðan<sup>148</sup> is the ordinary word for "to bind up, or to staunch".

Fumigation was known as a remedy for fever, &c. The word used is steran, styran, which meant to burn incense. Wio ðam fefore ðe ðu dryddan dæge on man becymed genim ðysse ylean wyrte twigu, befeald on wulle, ster hyne ðermid toforan ðam timan ðe se fefor hym to wyll<sup>149</sup> ("For a tertian fever: take twigs of this same herb, fold them up in wool, perfume him therewith before the time that the fever will again be upon him").

Gledfæt is found for a chafing-dish on which herbs for fumigation were laid. Do ðonne gleda an gledfæt ond lege ða wyrte on, gerec ðone man mid ðam wyrtum ær undern ond on niht ond sing letania ond credan ond pater noster ond writ him cristes mæl on ælcum lime<sup>150</sup> ("Then put red-hot coals in a chafing-dish and lay the herbs on it; smoke the man with the herbs before 9 a.m. and at night, and sing the litany and the creed and pater noster, and write the sign of Christ on each limb").

Tigehorn appears for "a cupping-glass".<sup>151</sup> A tooth-pick is recommended for putting an ointment into the eye.<sup>152</sup>

<sup>129</sup> Wrt. Voc. 67/8. <sup>130</sup> Lchdm. III. 22. <sup>131</sup> Shrn. 80/5. <sup>132</sup> Leon. 76/31. <sup>133</sup> Ib. 81/1. 9. <sup>134</sup> Ib. 81/10. <sup>135</sup> Ib. 79/39. <sup>136</sup> Ib. 51/25. <sup>137</sup> Leon. 143/26. <sup>138</sup> Lchdm. I. 354. <sup>139</sup> Ib. I. 76. <sup>140</sup> Ib. III. 134. <sup>141</sup> R. Ben. 138/2. <sup>142</sup> Leon. 66/13. <sup>143</sup> Ib. 4/6. <sup>144</sup> Ib. 22/27. <sup>145</sup> Wrt. Voc. II. 39. <sup>146</sup> Ib. II. 39/69. <sup>147</sup> Leon. 63/1. <sup>148</sup> Hmbo. T. II. 136/2. <sup>149</sup> Lchdm. I. 294. v. I. 98. <sup>150</sup> Leon. 105/25. <sup>151</sup> Ib. 37/17. <sup>152</sup> Ib. 12/11.

It is pleasing to find that the treatment prescribed in the Old English medical treatises sometimes, at least, failed to hinder recovery and sometimes even gave relief. *Se earpnumol* occurs several times for efficacious,<sup>153</sup> and *retan* is found for to relieve.<sup>154</sup> *Wierpe*, improvement, is found in the medical literature for convalescence,<sup>155</sup> and *edwierpung*<sup>156</sup> is found once with the same meaning. The verbs (ge) *wierpan*, *awierpan*, mean to recover, a process which did actually take place. *Weard him ða geduht swilce heo gewurpan* (v.r. *awyrpan*) *mihte*<sup>157</sup> ("It seemed to him then as though she might recover").

The references are the same as in Bosworth and Toller's *Anglo-Saxon Dictionary* and in Toller's *Supplement* to the Dictionary, published respectively 1882 and 1921, by the Oxford University Press. To them add PAYNE, *English Medicine in the Anglo-Saxon Times* by Joseph Frank Payne. Fitzpatrick Lectures, 1903, London; LEON, *Kleinere angelsächsische Denkmäler* by G. Leonhardi, being Band VI of R. P. Wülker's *Bibliothek der angelsächsischen Prosa*, Hamburg, 1905.

[December 6, 1939]

### Philip Syng Physick. 1768-1837

By GEORGE EDWARDS, M.R.C.S., L.R.C.P.

PHILIP SYNG PHYSICK was born in Philadelphia on July 7, 1768. His father, Edmund Physick, was Keeper of the Great Seal and Receiver-general of the Colony of Pennsylvania; after the Revolution he became agent for the Penn estate. Physick senior was anxious that his son should be a doctor, but the boy at first showed no particular inclination to this course. He graduated in the Faculty of Arts at the Pennsylvania University in 1785 at the age of 17. He also worked for some time with his maternal grandfather, Philip Syng, who was a silversmith of repute, and this early practical training is said to have borne fruit in Physick's later marked ability in the contrivance of instruments.

His father's desire that Physick should study medicine now prevailed and the young man attended the courses of instruction conducted by Adam Kuhn. Kuhn had been a pupil of Linnaeus, was Professor of Botany and *Materia Medica* in the University of Pennsylvania and was Physician to the Pennsylvania Hospital. It might have been expected that Physick would go on to medical qualification in Philadelphia, but in November 1788 he and his father sailed for England. In London they obtained an introduction to John Hunter who took the young man as a pupil. The story is frequently repeated that when the elder Physick inquired what books his son should read, Hunter led the way into the dissecting room, showed them the bodies and said, "These are the books your son will read under my direction: the others are fit for very little".

Physick's name appears on the students' roll at St. George's under the date May 11, 1789, and he is assigned as dresser to John Hunter for one year.

In December 1789, the weekly Board met and received young Mr. Physick on his appointment as House Surgeon for the coming year. The Board also received twenty-five pounds of Mr. Physick's money to pay for his board and lodging. It is generally stated and it seems most probable that Physick owed his appointment to Hunter's sponsorship. Randolph, Physick's son-in-law and biographer, says that no little ill-feeling was aroused among the unsuccessful applicants. Physick completed his year of office with confessed profit to himself and, if Randolph is to

<sup>153</sup> Lchdm. I. 134. <sup>154</sup> Leon. 108/6. <sup>155</sup> Ib. 15/24. <sup>156</sup> Hom. Th. II. 26/29. <sup>157</sup> Hom. Skt. I. 436/65.

be believed, to the expressed gratification of the hospital authorities. The only other direct reference to his work at St. George's is by Hunter in his *Treatise on the Blood, Inflammation, and Gun-shot Wounds* (Part I, Section vii), where we read: "Many of these experiments were repeated, by my desire, by Dr. Physick now of Philadelphia, when he acted as House Surgeon at St. George's Hospital, whose accuracy I could depend upon." At one time during this year Physick was seriously ill; the diagnosis is nowhere given, but his condition was so grave that Hunter recommended his immediate return to his home. This, however, did not prove to be necessary.

At the end of 1790 Physick is said to have received the Diploma of the Royal College of Surgeons. The College had not then been incorporated and Physick's name cannot be found in the roll of the Surgeons' Company. He spent a few more months with Hunter, but declined an offer to become the great surgeon's assistant. In May 1791 he departed for Edinburgh, then the spiritual home of the English-speaking medical world. Here, in 1792, he received his doctorate, presenting a thesis on *Apoplexy* in Latin and dedicated to Hunter. Returning to Philadelphia in September 1792 he put up his plate there at the age of 24.

Accounts differ as to the rapidity with which his practice developed, but he himself complained in later years: "I walked the streets of Philadelphia after my return from Europe for nearly three years without making as much by my practice as would put soles on my shoes."

Nevertheless the famous yellow fever epidemic of 1793 seems to have brought Physick into contact with the great Benjamin Rush. This leader of the medical profession was also a politician of renown and had been one of the signatories of the Declaration of Independence. Incidentally, he used on that occasion an inkstand made by Physick's grandfather Syng. Rush's doctrine of "disorders of nerve force", derived from Cullen of Edinburgh, was the most bruited idea of the time; but all his theory failed before the ravages of the yellow fever. Suddenly he became convinced that purgation and bleeding were the essential treatment for this fever: in fact, it was the treatment for all fevers, and indeed, for all morbid conditions. Physick put forward the view to which Rush assented much later that yellow fever was not contagious and he came to Rush's support in the inevitable argument as to the value of bleeding with a public announcement in the *Gazette* of the United States of November 14, 1797: "With a view to inspiring confidence in blood-letting in cases of yellow fever, I take this method of informing my fellow citizens that I lost during my last attack of that fever 176 ounces of blood by 22 bleedings in ten days. The efficiency of this valuable remedy was aided by frequent copious evacuations from my bowel and a moderate salivation." This seems to be a remarkable contribution to a public journal and the more so in that Physick was strenuously opposed to public expression of his views. It is recorded too that in his declining years Physick expressed regret not that he had bled his patients so much but that he had not used venesection more freely.

In 1794 Physick was elected to the staff of the Pennsylvania Hospital and to that of the Philadelphia Dispensary. His life began to be full and we must examine its various aspects separately. First we may take his qualities as a surgeon; they seem to have been excellent. He is said to have operated with firmness and precision and with reason. His private practice became extensive largely because of his success in operating for two somewhat different diseases. He was extremely successful in enucleating the lens from the eye and in removing stones from the bladder. The first recorded operation in his private notebook is one of lens enucleation and so was his last performed on August 13, 1837, the day on which he suffered the first attack of his final illness. The most notable of his bladder operations was on Chief Justice Marshall, from whom he removed over one thousand stones. The patient was of advanced age and great renown; the mere suggestion of the operation had caused

some discussion; but in spite of this untoward circumstance, both surgeon and patient played their parts in an exemplary fashion and the Chief Justice lived some further years freed from the agonizing pain of his multitudinous calculi.

When we turn to the various new surgical methods, new instruments and new lines of treatment which Physick improvised, invented or instituted we shall see how well deserved was his fame. Immediately on his appointment to the Pennsylvania Hospital he tackled the problem of chronic ulcers, and by instituting a régime of rest for the affected limbs, he rapidly reduced the numbers of bed-cases which were proving a heavy burden on the hospital's resources. He made many improvements in the treatment of fractures: particularly he lengthened Desault's splint for fractures of the thigh. For intractable dislocations, he conceived the idea of copious bleeding even *ad deliquum animi*; this so reduced the resistance that reposition was possible. He contrived a stomach pump on being called to treat two small boys who had drunk laudanum. Monroe II of Edinburgh had done this some years before, but Physick's invention was an independent one. He invented a special forceps and needle for controlling hæmorrhage by under-running the vessels. He designed various modifications of catheters, particularly the bougie-headed catheter. He constructed improved types of gorget for his lithotomy operations. He devised a double cannula with a wire loop for snaring tonsils and hæmorrhoids. He devised the tonsillotome.

He first achieved a successful operation "for the cure of artificial anus"—by this, I take it that he succeeded in closing a faecal fistula. He experimented with absorbable animal ligatures; successfully treated a non-united fracture of the humerus by a seton; put into operation the idea of immobilizing the joint in cases of hip disease—a line of treatment extended to other joints; gave his name to a special pair of dental forceps and introduced into America the Wenzel operation of lens enucleation.

All these activities deserve our respect but hardly justify his title as "Father of American Surgery", nor can his published writings earn him that distinction. He was extremely averse from putting his ideas into print and it is said that he even endeavoured to prohibit the posthumous publication of his notes. All that can be collected of his published works is a series of technical descriptions of new instruments and new methods.

It was in his ability as a teacher of surgery that Physick was most outstanding. In his earlier years in Philadelphia the Professorship of Surgery was combined with that of Anatomy and the surgical teaching was then much circumscribed. In 1800, Physick was approached privately by a number of students to lecture at the Pennsylvania Hospital. This he did, approaching the first lecture with natural diffidence and learning his discourse by heart. The lecture was welcomed and Physick continued his course, repeating it until in 1806 he was invited to a new separate Chair of Surgery. He took his lecturing with extreme seriousness; it was his practice to rise at 4 a.m., make his own fire and prepare his lecture notes before starting on the day's round. He had a complete mastery of his subject, but never went beyond the bounds of observable fact and of his own experience. The University of Pennsylvania owns several copies of notes made during his lectures. The actual discourses may have owed much to Physick's own style of delivery and this perhaps was his reason for refusing to publish them.

In 1819 he was transferred from the Chair of Surgery to the Chair of Anatomy, in which he succeeded his own nephew, John Syng Dorsey. This young man had early been apprenticed to his uncle, had qualified in Philadelphia and had, like his uncle, been a pupil at St. George's Hospital, but not until 1803, ten years after Hunter's death. Dorsey had practised in Philadelphia largely as Physick's junior and had in 1816 been appointed Professor of Materia Medica. In 1818 he was elected Professor of Anatomy and on November 2 he gave his inaugural lecture.

That same night he was attacked by typhus and died in less than a fortnight. For some reason of internal politics it was suggested that Physick should take over the Chair of Anatomy and relinquish that of Surgery. He went, in the words of one of his biographers, John Bell, "from the place where he was emphatically at home to one in which he was comparatively a stranger". "The act", says another, "was a descent from his high estate which dimmed and deadened his academic lustre."

The Chair of Surgery was filled by the appointment of the young William Gibson who, at 23, had been the first Professor of Surgery in the Maryland Medical School inaugurated in Baltimore in 1811. The two young men, Dorsey and Gibson, were the channels through which Physick's surgical teachings reached a wider world than that of his pupils and listeners. In 1813 Dorsey published *The Elements of Surgery* in two volumes. The book was well received in Edinburgh and was generally considered to represent Physick's teachings. A second edition appeared in Dorsey's lifetime and a third after his early death. Gibson published in 1824 *The Institutes and Practice of Surgery* and this, too, was said largely to derive from Physick's teaching.

In 1831, at the age of 63, Physick's failing health caused him to retire from active duties at the University. He was unanimously elected Emeritus Professor of Surgery and Anatomy "as a tribute to his merit in elevating the character of the school and in promoting the advance of medical science".

Physick was ill in London in 1790. He had two attacks which were said to have been yellow fever—one in the epidemic of 1793 and another in 1797. In 1813 he had "typhus". He had always suffered from catarrh and he was subject to repeated attacks of renal colic. His heart began to fail and in the summer of 1837 he developed hydrothorax. This caused such dyspnoea that for whole nights together he was unable to lie down. The enforced standing aggravated the increasing oedema of his legs till finally the overstretched skin broke down leaving gangrenous ulcers. He died on December 15, 1837.

Of Physick's personal characteristics we know little. Throughout the greater part of his life he is said to have lived lonely and alone. He used few words and he was impatient of verbosity in others. According to Randolph his colleagues and pupils admired him and his family were fond of him. For the most part we are left to assume that Physick could not suffer fools gladly, and that his bodily discomforts shortened his temper.

There is an unfortunate example of Physick's "common sense" in the story of McDowell's first paper on *Ovariectomy*. One of the first copies of this paper, which was written in 1817, was sent to Physick as the leading surgeon of the time. Physick dismissed with contempt the startling information contained in the crudely written and incomplete description. "The Father of American Surgery", says Flexner, "was too knowing to be taken in by a nonentity's crude description of the impossible". All that can be said in mitigation of this lamentable lack of imagination is that Physick did stop at condemnation. Many later surgeons were not only guilty of denying the value of McDowell's work in operating on ovarian tumours but they seem to have tried later to arrogate his glory to themselves.

Physick's appointments and honours were many. He was not only Physician to the Pennsylvania Hospital, but to the Philadelphia Dispensary. He seems to have been particularly pleased when the Pennsylvania Almshouses appointed him as "Surgeon Extraordinary". From 1824 to his death he was President of the Philadelphia Medical Society. In 1825 he had the distinction of being the first American to be appointed a member of the Royal Academy of Medicine of France. In 1836, the year before his death, he was accorded the honour which is said to have pleased him most—he was elected an honorary member of the Royal Medical and Chirurgical Society of London, the predecessor of the Royal Society of Medicine.

## Section of Orthopædics

President—T. P. McMURRAY, M.Ch.

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[December 5, 1939]

### Simplifications of Technique in Some Joint Operations

By M. FORRESTER-BROWN, M.S., M.D.

THE increase of bone and joint disabilities, due first to motor accidents and now to war, makes it desirable that simple procedures, which can be carried out by relatively inexperienced surgeons, should be as widely known as possible.

#### *A. Sliding Osteotomy for the Correction of Joint Deformities and Severe Contractures*

The principle of this method is that the bone, either in a joint which is ankylosed or adjacent to a mobile joint should be divided in its long axis with enough obliquity for the fragments to glide on one another and so to shorten the limb till the desired tension on the contracted soft structures is reached.

The following are applications of the method :—

(1) *Flexed knees* in which the capsule is so much shortened that dangerous tension would be put on the popliteal nerves and vessels if it were cut. If bony ankylosis is present, the section is made through femur and tibia from above and without downward and inward in a sagittal plane. If the joint is movable, the lower third of the femoral shaft is divided in the same plane. The tight hamstrings make the femoral fragments telescope till the knee is just straight.

(2) For malunited *Pott's fracture*, usually with valgus deformity and short peronei, the tibia is cut in the coronal plane just above the ankle-joint, and the fibula higher, e.g. at the level of original fracture. The malleoli and foot are slid inward on the leg till the ankle line is horizontal. This applies to an old neglected case with non-reduction in more than one plane, not to one that can be re-broken at its original level.

(3) *Hallux valgus* due to congenital metatarsus varus; the first metatarsal is split from its inner side downward and outward, so that as the great toe retracts, its tip moves inward.

It may be pointed out that the true Lorenz osteotomy for dislocated hips (coronal intertrochanteric osteotomy of femur) follows the same principle.

The advantages over wedge osteotomy and plastic operations on the soft parts are: (1) Avoidance of danger to the circulation from tension on vessels, and from dissection; (2) avoidance of the difficulty of judging the correct amount of wedge; (3) avoidance of the risk of overshortening and non-union; (4) the provision of large surfaces of raw bone in close contact, and hence rapid union, even in a devitalized limb.

Sliding osteotomy can also be used to balance inequalities of growth in the two leg or forearm bones, whether congenital, or due to epiphyseal injuries.

#### *B. Arthrodesis by Drilling the Articular Surfaces of a Joint*

The idea of this method arose from noting the rapidity of union in rickety bow-legs in which, before moulding, the bone had been softened by drilling multiple holes at the summit of the curve. As joint cartilage proves itself an ossifying tissue in many diseases, it seemed likely that, if enough bone cells were let loose in it, bony ankylosis would follow. Experience has shown this to be so, and the method has proved useful in the following conditions:—

(1) *Tuberculous knees* in subjects with active lung disease in whom a general and long anæsthetic is contra-indicated, as also is any extensive opening up of infected tissues.

One patient, with multiple sinuses and constant tubercle bacilli in the sputum, not only got rapid bony union with relief of pain, but also in due course healing of all sinuses and absence of sputum.

(2) *Claw-toes*.—After the opposing articular cartilages of one interphalangeal joint, or both, have been broken up by a fine awl, the toe can be stabilized by a strong silkworm gut stitch passed longitudinally through all its tissues and tied so as to hyperextend the interphalangeal joints and hold the raw areas in contact.

A comparable operation was tried on one child of 2 years with a flail shoulder due to poliomyelitis and, though only fibrous union was obtained, this stabilized the limb sufficiently for her to dispense with a shoulder splint and get increasing return of power in the elbow and forearm muscles. A formal arthrodesis later should prove easy because the maintenance of apposition between the articular surfaces must prevent the formation of an hour-glass constriction in the capsule, such as otherwise tends to occur in unsplinted cases.

#### *C. Arthrodesis of Flexed Joints by Removal of a Wedge from the Convexity*

This is particularly applicable to rheumatoid knees and wrists, contracted in flexion, in which loss of cartilage has caused permanent restriction of mobility and a useless and painful range. The patients are unsuited to long operations and extensive manipulations, and a knee or wrist can be fixed in good position in a few minutes by a skin incision across the convexity and the removal of a small wedge from both bone ends. The friable bone can then be crushed till the required position is attained, such compression inducing rapid new bone formation. A plaster slab splint can be rapidly applied. Pain is negligible after such an operation, so that these weakly patients are not devitalized by shock and loss of sleep. Walking in a caliper can be allowed in three or four weeks.

*D. Sliding Graft from Malleolus for Tuberculous Foot*

Many tuberculous feet can be saved by conservative treatment in a closed plaster to include the knee until fibrous ankylosis has occurred; then, to prevent relapse from strain, all the affected joints should be arthrodized by a bone graft. Many complicated operations have been devised for this purpose. I have found a simple procedure quite effective. An incision is made on the less damaged side of the ankle and foot, and with a gouge, a flap of bone and periosteum is raised from the tip of the malleolus downward till the distal limit of the diseased tissue seems to have been reached. The width of the strip is such as will open all the affected tarsal joints. Any caseous or devitalized tissue can be excised. A graft is cut of corresponding length and breadth from the exposed malleolus and shaft above it. The graft is turned down, and impacted with a punch into the soft tarsus. As much of the flap as is convenient is sutured over the graft. A plaster, applied so as to include the knee, is retained for about three months, or until X-rays show some trabeculae crossing the main joints. Then the patient walks in a leather mould until the X-ray shows firm bony union. After this he wears an ordinary shoe with a cork wedge in the heel, because the foot is grafted in slight equinus to maintain its spring; it is also put in moderate valgus. Although this treatment demands a longer period in hospital than amputation, this is an advantage, because it enables the primary focus from which the foot was infected to heal; it also dispenses with all apparatus once healing has occurred and the stiff living foot has the advantage over the artificial foot that sensation is retained.

*E. Sliding Graft from Scapular Spine for Tuberculous Shoulder*

A similar operation is applicable for a quiescent tuberculous shoulder, which has been got into good position by splinting, i.e. about 30° flexion, 30° external rotation, and 70° abduction. It is best to start with this degree of abduction, because in these cases the callus remains soft long after bony union is indicated by the X-rays, and the patient has felt comfortable without a splint, so that one always ends with less abduction.

A curved incision is made over the back of the shoulder with the patient lying half-prone, tilted towards the sound side. The posterior half of the deltoid is detached from its origin and retracted forward; then the capsule and attachments of the spinati are split and retracted with a sharp periosteal elevator. A bed for the graft is dug in the upper third of the humerus and the back of the glenoid, if that can be reached without opening much tuberculous tissue. Then with a gouge the acromion and sufficient of the scapular spine is detached and slid outward to form a bridge from the base of the spine to the great tuberosity. The deltoid is sutured back under tension to fix the graft, and an abduction plaster is applied.

*F. Sacro-iliac Arthrodesis by Wedge Graft*

A skin flap is raised from the back of the sacrum and the aponeurosis turned outwards till the posterior aspect of the joint is exposed. A thin wedge is removed from it, and a graft from the neighbouring iliac crest is impacted in the gap. The aponeurosis and skin are sutured over the graft.

[NOTE.—This paper was illustrated with a number of convincing skiagrams.]



FIG. 1.



FIG. 2.

Case I.—Position of fragments, one and a half years ununited.

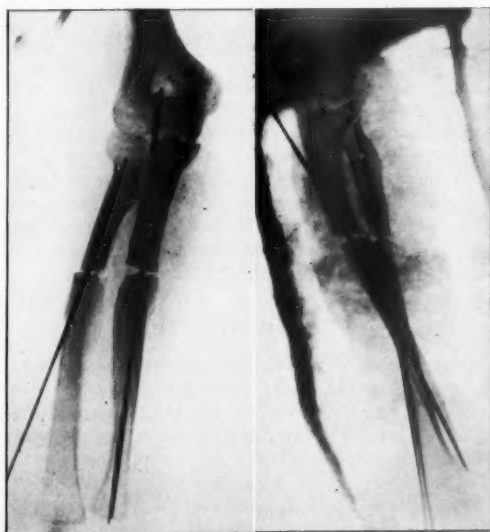


FIG. 3.—Position of fragments after freshening the ends and inserting wires.  
In this case the wires were brought out through a window in both bones.

(Illustrating Mr. Lambrinudi's paper, p. 17.)

## Intramedullary Kirschner Wires in the Treatment of Fractures

By C. LAMBRINUDI, F.R.C.S.

THE use of intramedullary wires in the treatment of fractures has, so far as I know, not previously been described. I have tried it three times with success.

The first case was of ununited fracture of the radius and ulna of one and a half years' duration. The application of a bone graft seemed the obvious treatment but, being an indifferent carpenter, I hesitated to undertake the grafting of these two bones at one sitting, for it is an operation which I have always found difficult and by no means always successful, even in the one bone. All I did was to freshen the ends of the fragments, insert the wires, apply plaster, remove the wires at the end of five weeks, and retain the plaster for several months. Eventually union occurred, the radius by consolidation, the ulna by periosteal bone; and though the position was poor, the result indicated that this method of immobilization and apposition was good.

(See Figs. 1, 2 and 3 opposite, Fig. 4 below.)



Case 1, FIG. 4.—Final result fifteen months later. Note.—The fragments have not slipped, union has occurred, but the position is not good because the upper fragment of the radius is pronated and the lower fragment is supinated. I had not observed that the upper fragment after two years of non-union was fixed in pronation, and failed to put the lower fragment into alignment with it.



Case II, FIG. 1.—Bad position after six weeks. Cross union beginning to occur.

The second case was almost identical so far as the position of the fragments was concerned. The fracture was comparatively recent (six weeks), but cross-union was beginning to occur. I merely inserted the wire without freshening the ends of the fragments, and applied a supination plaster. The wire was removed at the end of four weeks, and now, after seven weeks, the radius feels firm but there is still some give in the ulna. Delay in union always occurs after open operation on fractures, but I have little doubt that solid union will occur in time. (Figs. 1, 2 and 3.)

The third case is an even more convincing demonstration of the value of this method. A boy aged 10 had an ankylosis of the hip, the result of infection, with the limb fixed in external rotation and abduction. His walk was ungainly and he was developing scoliosis. I dared not make the correction at the usual site for fear of recrudescence of the disease; so I decided to make it at the middle of the shaft of the femur. I cut a window low down in the femur, passed some Kirschner wires up the shaft, did the osteotomy, corrected the external rotation, and gave him the requisite amount of bowed femur to compensate for the abduction of the hip. The wires were removed at the end of six weeks and the plaster retained for four months. The X-ray photographs show that by no other method could one's objective have been obtained so well and easily. (See p. 20, figs. 1, 2 and 3.)

The technique is simple. In the case of the forearm the site of fracture in the ulna is first exposed, the wire inserted into the upper fragment up to the olecranon and through the skin. It is then pulled up, the lower fragment placed in alignment and the wire pushed down into it, leaving 2 in. protruding from the olecranon. The radius is a little more difficult in so far as there is no free end to drill the wire through, but the difficulty is overcome by making a window with the electric saw



Case II, FIG. 2.—After insertion of wires. Note that the wire in the ulna is extruding from the olecranon.



FIG. 3.—After seven weeks. The wires had been removed three weeks previously.



Case III, FIG. 1.—Note abduction of hip. The 90° external rotation is not demonstrated.



FIG. 2.—Position after insertion of wires through a window and osteotomy. Note the amount of bowing required to compensate for the abduction of the hip. External rotation completely corrected.  
A.—Lateral view. B.—Antero-posterior view.



FIG. 3.—After four months. Note solid union; the fragments have not slipped.

in the longer fragment remote from the site of fracture. The wire is then passed into the medulla from the fractured end and pulled out to the requisite amount through the window, by means of a loop of salmon gut or silver wire; the other fragment is placed in position and the wire pushed into it, leaving 2 in. protruding through the window and wound. A supination plaster is then applied incorporating the wires.

This method will be found to have its uses but it also has its limitations. It is designed to prevent lateral displacement, but if the fracture is transverse it also prevents shortening, for at least half the diameter of the fragments must always be in apposition, and the splint controls angulation. In a long spiral fracture the method prevents lateral displacement only, and cannot prevent telescoping. It is, however, applicable to fractures of two bones if one of them is fractured transversely, for, if this latter is stabilized by means of a wire, the fragments cannot become displaced; and the other bone, which is fractured spirally, cannot telescope without angulation. Fortunately, in fractures of the radius and ulna one of them, usually the ulna, is fractured transversely, and a well-applied plaster prevents angulation.

### **Treatment of Ununited Fractures by Bone Grafting without Resection of the Bone Ends**

By H. JACKSON BURROWS, M.D., F.R.C.S.

THIS paper is confined to the consideration of one method of treatment of a single type of case of non-union of a fracture of the shaft of a long bone—that in which there is a fibrous intersection rather than a really wide gap between the fragments.

The treatment under investigation is that of autogenous bone grafting with minimal disturbance of the bone ends or the intervening tissue. It is usually advised that a graft operation should be accompanied by clearing and resection of the bone ends. Some surgeons remove all sclerotic bone; others are more conservative, and the procedure is then sometimes referred to as "freshening" the bone ends. (Mr. Naughton Dunn has used the same term for his highly successful subcortical procedure, but this involves no removal of bone and does not therefore enter into the considerations of the present paper.) It has long been Mr. R. C. Elmslie's teaching that the customary resection of the bone ends as a supplement to grafting is unnecessary and therefore undesirable. To test the validity of this contention the results of a consecutive series of cases treated by Mr. Elmslie and two of his pupils have been investigated and are here reported. Unfortunately no series of control cases treated by the more extensive procedure is available, because this has not been used by us in recent years.

In the cases under review the graft adopted was an osteo-periosteal inlay from the tibia, except in two instances: an intramedullary tibial graft to the humerus, and a fibular peg to the femoral neck. Tibial grafts were always taken from the uninjured tibia and never from the fractured one. In operations on the leg, osteoclasis or osteotomy of the fibula was performed only if necessary for good general alignment.

There are difficulties in assessing the results of any method of treatment for non-union. First there is the relative scarcity of cases; I have only 17 of the type under discussion to report. Secondly the inadvertent inclusion of cases of mere delayed union may give an unduly good impression of the results of operation. The present series (see Table I) includes six cases in which operation was performed less than a

year after fracture. Some of these cases might perhaps ultimately have united with further conservative treatment, although no case was operated upon in which this was considered reasonably likely; on the contrary, selection was made in a conservative spirit.

Bony union was obtained in all cases except one, in which there was no evidence of union at the end of fifteen months, when the patient defaulted; almost certainly this case was a failure. The probable cause for this was that plaster-of-Paris immobilization was discontinued only eight and a half weeks after operation, because the fracture felt firm, and an ineffective appliance was substituted.

The time taken for union to occur in the successful cases was usually four to five to eight months, but in two cases periods of respectively nine and thirteen months passed before union was assuredly present. The figures should be accepted with reserve, because the assessment of time of union is difficult. In the lower limb a further period of protection is required after union has become established.

Two cases had previously been operated upon unsuccessfully for non-union by drilling. Two complications (both in cases of the writer) occurred—staphylococcal infection of a superficial hæmatoma in the oldest patient, and cellulitis, eight months after operation, in another. In neither case was there any serious consequence.

The results of the simple grafting operation are dependent on the method itself and not on the special technical skill developed by one surgeon, because the series is collected from cases operated upon by three different surgeons (see Table III).

With the reservation required in deducing from a necessarily small series of cases, it is concluded that, in the operative treatment of non-union unassociated with wide separation, a grafting operation need not be accompanied by the customary freshening and resection of bone ends. If this procedure is unnecessary, it should be discontinued as a supplement to grafting because: (a) It adds to the duration and severity of the operation, and therefore to the likelihood of complications. (b) It causes either shortening or the risk, should the operation fail, of a flail limb. (c) The additional stripping must interfere with the blood supply. (d) The most precarious cases for grafting are those in which either the original accident or Nature has produced a gap which requires bridging; just such a condition may be produced by the surgeon who performs a resection. (e) In the case of fracture near the end of the shaft one fragment is already undesirably short for holding an adequate extent of the graft.

#### SUMMARY

(1) Seventeen consecutive cases of ununited fracture of a long bone treated by grafting are described, the cases being those without a wide gap requiring bridging; the operation in each instance was the application of a bone graft without resection of the bone ends.

(2) With due regard to the relative smallness of the number of cases, the results suggest that resection of the bone ends is an unnecessary supplement to the application of a graft.

(3) Reasons are given for supposing that resection is not merely superfluous but actually harmful.

I have to thank Mr. R. C. Elmslie and Mr. S. L. Higgs for permission to include their cases. My thanks are particularly due to Mr. Elmslie, not only for the inception of this paper, but also for allowing me to operate upon certain patients under his care.

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TABLE I.—SUMMARY OF PRINCIPAL FEATURES OF THE SERIES OF CASES. THEY ARE ARRANGED IN ORDER OF THE DURATION EACH FRACTURE HAD BEEN PRESENT BEFORE OPERATION.

Case	Sex	Age	Bone fractured	Mos. since fracture	Mos. for union after op. (approx.)	Occupation before accident	Occupation fully resumed after op.	Mos. of incapacity after graft op.	Remarks
J. Pn.	F	32	Tibia	5	8	Housewife	The same	13	Resumption delayed by cellulitis, which occurred 8 months after operation
D. G.	M	28	Humerus	5	8	Bus conductor	General hand in small garage	20	Arthroplasty of elbow also. Found the work too heavy and changed it, after 12 months, for petrol storeman
R. H.	M	18	{ Radius Ulna	6 6	5 5	Carpenter	The same	7	
A. F.	F	61	Tibia	9	6	Housework	(Housework exclusive of kneeling)	19	The fracture was at the upper end of the tibial shaft
E. V. F.	M	±27	Ulna	10	(?)	Nil	(Untraced)	(?)	Union known to have occurred
J. B.	F	18	Femoral neck	12	9	Clerk	Shop assistant	9	
T. L.	M	39	Radius	14	4	Market porter	Casual market porter	12	Unable to do heavy work on account of pulmonary tuberculosis
C. W.	M	10	Radius	15	6	School	The same	10	The figure is for full resumption, with games
T. T.	M	23	Tibia	15	6	Motor mechanic	Shop cleaner	12	
T. N.	M	32	Tibia	15	13	Lorry driver	The same	21	
E. P.	F	24	Tibia	17	6	Secretary	The same	6	
R. K.	M	21	Tibia	22	4	Outdoor salesman	The same	9	
A. L.	M	41	Tibia	22	8	Shop assistant	The same	15	
J. Pr.	M	22	Tibia	28	3	Dairyman	The same	6	Subsequently took sole charge of business; 12½ hours active work daily; is also a special constable
T. R.	M	23	Tibia	30	Not united after 15 mo.	Machinist	(Untraced)	(?)	
P. W.	F	14	Metatarsal	60	5	School	The same	8	The figure is for full resumption, with games

TABLE II.—SUMMARY OF THE RESULTS OF OPERATION FROM THE VIEWPOINT OF ITS PRIMARY OBJECT.

<i>Results</i>			
Number of cases grafted	..	..	17
Number followed by bony union	..	..	16
Failure ..	..	..	1

TABLE III.—DISTRIBUTION OF CASES IN THE PRESENT SERIES AMONG DIFFERENT SURGEONS. THIS TABLE SHOWS THAT THE RESULTS DEPEND ON THE METHOD AND NOT ON THE SPECIALLY DEVELOPED TECHNICAL SKILL OF AN INDIVIDUAL OPERATOR.

Operating surgeon			No. of patients operated on	No. of bones receiving graft
I	..	..	7	7
II	..	..	2	3
III	..	..	7	7
			<hr/> 16	<hr/> 17

## Section of Comparative Medicine

President—C. H. ANDREWES, M.D., F.R.S.

[December 13, 1939]

### DISCUSSION ON NERVOUS AFFECTIONS IN MAN AND ANIMALS

**Dr. G. M. Findlay**, *Wellcome Bureau of Scientific Research, London*: Broadly speaking, the reactions of the central nervous system to injury are similar in man and animals. Nevertheless, there are many differences in the character of the diseases peculiar to man and animals, and in the degree of reaction.

Diseases such as the hereditary ataxias or amaurotic family idiocy are unknown in animals; hereditary tremor, however, has been recorded in rabbits as well as a disease resembling syringomyelia which has been observed through eight generations (cf. Frauchiger, 1936). Though congenital hydrocephalus is not uncommon in animals and man, there is nothing in human pathology corresponding to "Dummkoller" of horses, a disease believed to be an inherited hydrocephalus.

Degenerative vascular lesions are less frequent in animals than in man while tumours, particularly gliomata, are said to be comparatively rare. Great differences are seen in the reaction of the central nervous system to certain poisons; the extreme sensitivity of the dog to strychnine is well known. In man, chronic manganese poisoning induces a specific degeneration of the lenticular nucleus and rarely cirrhosis of the liver. In rhesus monkeys and small rodents, on the other hand, liver cirrhosis is very easily produced by manganese, but no changes are found in the lenticular nucleus. There are also differences in the relative susceptibility of the central nervous systems of man and animals to parasitic infections. While toxoplasms cause encephalitis in man and animals and *Leishmania donovani* is responsible in the Mediterranean area for meningitic symptoms both in children and in dogs, the numerous blood trypanosomes of animals do not invade the central nervous system, and among animals there is nothing corresponding to the nervous stage of human sleeping sickness caused by *Trypanosoma gambiense* and *T. rhodesiense*. Bacteria and spirochaetes also invade the central nervous system of man more readily than that of animals. Thus in animals there is no spontaneous bacterial infection corresponding to meningococcal meningitis nor under natural conditions do such organisms as *Pneumococcus*, *Streptococcus*, *Brucella*, *Salmonella*, or *Hæmophilus influenzae* readily attack the meninges of animals. In fact the only bacteria which naturally infect the meninges of both man and animals are *Mycobacterium tuberculosis* and *Listerella*. *Spirochaeta cuniculi* and *S. gallinarum* do not produce diseases of the central nervous system in rabbits and hens in any way analogous to human cerebrospinal syphilis, and while *Leptospira icterohæmorrhagiae* and *L. canicola* may occasionally cause meningitis in man, *L. canicola* has not yet been recorded as causing meningitis in the dog. There is thus a greater susceptibility of the human central nervous system to trypanosomes, bacteria, and spirochaetes; on the other hand animals and man are equally susceptible to a number of spontaneous virus infections.

#### VIRUS DISEASES OF ANIMALS AND MAN

The spontaneous neurotropic virus diseases may be divided into three groups: (i) viruses pathogenic only for man, (ii) viruses pathogenic only for animals, (iii) viruses pathogenic both for animals and man.

The first group includes zoster, St. Louis encephalitis, Japanese B type encephalitis and the encephalitides sometimes associated with mumps, herpes simplex, dengue,

and possibly varicella. Poliomyelitis must be included, although two spontaneous cases have been recorded in chimpanzees living in a Zoological Garden. Australian X disease may possibly fall into this group, unless like louping ill it is primarily a disease of sheep. With the exception of zoster all these viruses have been transmitted to laboratory animals, but there is no evidence to suggest that animals play any part in their epidemiology. It would not be surprising if evidence were brought forward to show that St. Louis encephalitis or Japanese B type encephalitis are really enzootic diseases only occasionally transmitted to man.

The second group, comprising neurotropic viruses normally pathogenic only for animals, includes fox encephalitis, mouse encephalitis, and probably Borna disease and swine fever. No evidence is yet available as to whether the Russian form of equine encephalomyelitis occurs naturally in man. Encephalitis probably of virus origin has also been found in cows and pigs and in the brains of such animals as polar bears and Canadian lemmings.

TABLE I.—NEUROTROPIC VIRUSES NATURALLY INFECTING ANIMALS AND MAN.

Disease	Animal hosts	Remarks
Rabies	Dogs, wolves, hyenas, jackals	
	Vampire bats	Symptomless carriers
Pseudo-rabies	Pigs, cattle	Infections only recorded in veterinarians
Louping ill	Sheep, horses	Infections only in laboratory workers
B. virus	Rhesus monkeys	Infections only in laboratory workers
Swineherd's disease	Pigs	Virus in faeces and urine of swine
Lymphocytic choriomeningitis	Mice	Virus in urine of mice
Equine encephalomyelitis	Horses, ring-necked pheasants, pigeons	Aedine mosquitoes may act as transmitters

The third group of neurotropic viruses (Table I) attacking both man and animals under natural conditions is an ever-growing one and at present includes rabies, pseudo-rabies, equine encephalomyelitis (both the eastern and western form) swineherd's disease, louping ill, and lymphocytic choriomeningitis. B. virus must also be included in this group, though there is no evidence that under normal conditions it attacks the central nervous system of monkeys.

A fourth group might be formed from those viruses which do not normally attack the central nervous system but which either spontaneously or, as a result of laboratory procedures, may have their neurotropism increased. This group would include vaccinia, yellow fever, African horse sickness, Rift Valley fever, influenza, fowl pest, and virus myxomatosis, which have been modified experimentally, and fowl pox which has undergone spontaneous variation. A number of other viruses which do not usually reach the central nervous system are capable, if injected intracerebrally, of giving rise to nervous lesions. Such viruses include the salivary gland viruses of guinea-pigs and mice and virus III of rabbits. Lymphogranuloma inguinale very occasionally causes nervous symptoms in man.

#### DEMYELINATING DISEASES IN ANIMALS AND IN MAN

If an attempt be made to classify the numerous demyelinating diseases according to what is now known of their aetiology, it is found that they fall into four groups associated with:—

- (1) Nutritional deficiencies.
- (2) Viruses.
- (3) Toxins.
- (4) Unknown aetiology.

In the group of demyelinating nutritional deficiencies found in man must be included pellagra and the subacute combined degeneration found in pernicious anaemia, while in animals there is swayback in lambs, associated with copper deficiency,

and the demyelinating lesions associated with vitamin-A deficiency. Whether any elements of the vitamin-B complex are responsible for demyelination appears doubtful. In the second group associated with virus infections are included Australian X disease which causes focal demyelination associated with inflammatory foci in the brain of man but not of animals (cf. Cleland and Campbell, 1919; Perdrau, 1936) and equine encephalomyelitis, both the eastern and western forms of which produce similar focal demyelination in man and animals (King, 1938; Branch and Farber, 1939; Breslich, Rowe and Lehman, 1939). The association of demyelination with equine encephalitis was first mentioned, however, by Dexter (1903). In acute distemper both in dogs and other *Canidae*, wolves, hyenas, jackals, small foci of demyelination have been found, more especially in the olfactory lobes, by Posrednik (1930), Marinesco *et al.* (1933), Peters and Yamagiwa (1935), and Hammerton (1937).

A series of post-infectious conditions has been grouped together as disseminated encephalomyelitis, Westphal's disease, or acute perivascular myelinoclasia (Marsden and Hurst, 1932). Acute perivascular myelinoclasia most commonly occurs as a sequel to vaccinia, variola, measles, and less frequently, varicella, and rubella (Bénard, 1921; Taylor, 1937; Davison and Friedfeld, 1938) and possibly after influenza (Greenfield, 1930). It has been seen after immunization against rabies and once after immunization against yellow fever (Lhermitte and Fribourg-Blanc, 1936). Paulian (1932) has also recorded a fatal case following experimental inoculation with the virus of herpes simplex. In dogs demyelination has been found in association with the acute stages of what has passed for distemper or as a later sequel (Perdrau and Pugh, 1930; King, 1939<sup>1</sup>) and also after immunization against rabies.

The third group includes ergotism and probably lathyrism in man, the subacute combined degeneration associated with alcoholism, and the demyelinating encephalitis associated with such drugs as arsphenamine (cf. Russell, 1937), sulphanilamide (Fisher and Gilmour, 1939) or uleron (Schubert, 1938). Demyelination has been produced experimentally in animals by a number of substances such as guanidine (Rosenthal, 1913), dysentery toxin (Lotmar, 1913), tetanus toxin (Claude, 1897; Putnam *et al.* 1931), staphylococcal toxin (Orr and Rows, 1918), maize infected with *Aspergillus fumigatus* (Ceni and Besta, 1905), potassium cyanide (Ferraro, 1933; Rubino, 1935), saponin (Donaggio, 1925), vinilamine (Luzzato and Levi, 1932), bile (Weil and Crandall, 1932), carbon monoxide (Grinker, 1925), and aqueous and alcohol-ether extracts of sterile normal brain (Rivers and Schwentker, 1935).

The fourth group includes in man disseminated sclerosis, Schilder's encephalitis, neuromyelitis optica, acute toxic (non-suppurative) encephalitis (Low, 1930), multiple degenerative softening (Hassin and Bassoe, 1922).

In animals, non-suppurative demyelinating diseases are found in primates, whilst demyelination may occur in dogs apart from distemper. King (1939<sup>2</sup>) has described a demyelinating disease in the moose apparently unassociated with any acute infective condition, and Hammerton (1936) observed a similar condition in the leopard *Felis pardus*.

The demyelinating disease of monkeys, frequently termed "cage paralysis", was first described by Rothman (1906) in Berlin. Since then a considerable number of cases have been reported in monkeys kept either in zoological gardens or under laboratory conditions in Europe (Schröder, 1908; Steiner, 1917; Levaditi, Lépine and Schoen, 1930; Perdrau, 1930; Schob, 1931; Scherer, 1932 and 1937; Gärtner, 1933; Levaditi, Hornus and Schoen, 1931, 1933<sup>1</sup> and <sup>2</sup>; Hammerton, 1936, 1937, and 1938; and van Bogaert, 1939), in America (Davison, 1934), and South Australia (Hurst and McLennan, 1937). The disease has been seen in *Simia satyrus*, Orang-utang (Schob), *Papio hamadryas* (Scherer, 1932; Gärtner), *P. cynocephalus* (Davison), *Macaca mulatta* (Steiner; Perdrau; Levaditi, Hornus and Schoen; Scherer; Hurst and McLennan and van Bogaert), *Macaca sinica* (Hammerton), *Erythrocebus patas* (Hammerton), *Cynopithecus maurus* (Gärtner), *Cercopithecus ascagnus* (van

Bogaert), *C. nictitans* (Hammerton and van Bogaert), *C. petaurista*, *C. ouanderou* (van Bogaert), *C. albogularis* (Hammerton), *C. albogularis mossambicus* (Hammerton), and a lemur *Maki catta gomböz* (van Bogaert). The disease is insidious in onset and retrogression of symptoms may occur so that the lesions are only discovered post mortem; nevertheless, the condition is usually progressive, and the various clinical types, optical, cerebral, medullary, and spinal are all forms of the same disease. The condition in monkeys has been compared to two human diseases, Schilder's diffuse periaxial encephalitis and neuromyelitis optica, but according to van Bogaert (1939) the lesions more closely resemble those caused in man by pernicious anaemia. This resemblance becomes closer when it is recalled that in some human cases of pernicious anaemia the white matter of the centrum ovale has been attacked (Bodechtel, 1937). In pernicious anaemia also a cerebral form with large multiple foci of demyelination has been described (Sjövall, 1937), while cerebral forms with progressive hemiparesis and psychical derangements, a medullary and a combined medullary and optic type, have been noted (Cohen, 1936).

Clinically and pathologically, therefore, the demyelinating disease of monkeys has close similarities with the nervous conditions found in pernicious anaemia, conditions that can be cured by the administration of a crude liver extract. The similarity to Schilder's encephalitis of swayback in lambs, the probable result of a copper deficiency (cf. Bennetts and Chapman, 1937) has already been emphasized by Innes (1936 and 1939). The possibility, therefore, suggests itself that Schilder's encephalitis, optic neuromyelitis and the nervous lesions due to pernicious anaemia in man, swayback in lambs, and the demyelinating disease of monkeys all resemble one another in being primarily due to a nutritional defect, arising either because of absence of certain compounds in the diet, defective absorption from the intestine, or defective formation of internal factors.

The evidence for a nutritional defect in the causation of cage paralysis of monkeys is as follows. The disease often occurs among small groups of animals (Levaditi *et al.*, 1933<sup>3</sup>; Schob, 1931; Gärtner, 1933; Scherer, 1932, 1937); this might well suggest a parasitic agent as the cause but although Gärtner claims to have transmitted the disease to normal monkeys, he gives no details, and in only one of five monkeys inoculated by Levaditi, Hornus and Schoen (1933<sup>1</sup> and <sup>2</sup>) did any symptoms result while the lesions in this one monkey differed from those in the spontaneously affected animals. The disease is afebrile, usually insidious in its onset, with improvements and relapses. Evidence in favour of insufficient nutrition is presented in two of the cases reported by van Bogaert (1939), while the monkey described by Perdrau had been badly treated by its companions. Dysenteric symptoms are not infrequent, and *Macaca mulatta* is often a carrier of *Bact. dysenteriae* (Flexner); when placed on a deficient diet, acute dysenteric symptoms may develop. Verder and Petran (1937) record dysentery in rhesus monkeys on a vitamin-A deficient diet, Topping and Fraser (1939) on a diet lacking flavin and nicotinic acid, Janota and Dack (1939) on a diet deficient in a portion of the vitamin-B complex which they term vitamin M. The toxins of *Bact. dysenteriae* were shown by Lotmar (1913) to be capable of causing demyelination in animals, whilst Langston, Darby, Shukers and Day (1938) found that anaemia and leucopenia may develop in monkeys on a diet lacking vitamin M. Few observations have been made on the blood changes associated with cage paralysis although Hammerton (1938), in a toque monkey, *Macaca sinica*, found that the haemoglobin was 80% and that the red-blood corpuscles showed pronounced poikilocytosis and anisocytosis. In a Mozambique monkey *Cercopithecus albogularis mossambicus* the haemoglobin was 80%, but the red blood corpuscles were normal in size and shape. This monkey had diarrhoea, while a patas monkey, *Erythrocebus patas*, had complete achlorhydria. Finally, in a Stair's monkey, *Cercopithecus albogularis stairsi*, symptoms highly suggestive of cage paralysis were cured by injections of campolon, a liver preparation, the number of red-blood corpuscles rising

from 4,200,000 to 5,800,000 per c.mm. Col. Hammerton (1939) kindly allows me to state that recently he has cured a second case of cage paralysis in a patas monkey by injections of campolon. Blood examinations made when the paralysis was very severe showed that it was not associated with either a micro- or macrocytic anæmia. The evidence, fragmentary as it is, suggests that cage paralysis is associated with a lack of some dietetic factor, and that it may be intensified by the presence of bacillary dysentery. Whether the deficiency is due to an extrinsic factor of unknown composition, as in pernicious anæmia, a trace element as in swayback, or a vitamin, further observations may determine. It is, however, of interest to note that Verder and Petran (1937), by feeding rhesus monkeys on a diet deficient in vitamin A, have produced blindness, fits and paralyses, all symptoms which have been noted in monkeys suffering from cage paralysis. Verder and Petran state, however, that they were unable to find any degenerative lesions in the central nervous system to account for these symptoms. Unfortunately, they do not indicate the extent or character of their histological examinations. In the cases described by Hammerton large doses of vitamins A, D, and B, failed to produce improvement.

Since the demyelinating syndrome in monkeys resembles in certain instances Schilder's encephalitis and neuromyelitis optica in man, the further question arises whether these conditions also may not be associated with a nutritional deficiency. All that can be said is that Schilder's encephalitis and neuromyelitis optica are not infrequently familial diseases (cf. Meyer and Tennent, 1936), that the onset is sometimes associated with diarrhoea or other intestinal derangements, and that Hurst and his colleagues (1939) have emphasized their comparative frequency in Southern Australia.

In discussing the primary myelinoclasts, it must first be emphasized that all cases of encephalitis occurring in association with virus infections are not necessarily characterized by demyelination. Encephalitic symptoms may in fact occur in the following relation to virus infections:—

- (I) Viruses may be predominantly neurotropic—rabies, poliomyelitis, louping ill.
- (II) Viruses not normally neurotropic may suddenly mutate and produce encephalitis—fowl pox (Buddingh, 1938).
- (III) Viruses not normally neurotropic may produce encephalitis if confined to the central nervous system by the intraperitoneal inoculation of immune serum (yellow fever, Rift Valley fever, fowl pest; Findlay and Stern, 1935, Mackenzie and Findlay, 1936).
- (IV) If repeatedly transmitted by intracerebral inoculation, certain viruses which normally produce viscerotropic lesions may gradually lose this power, and acquire the capacity to produce neurotropic lesions (yellow fever, Rift Valley fever, African horse sickness, influenza).

In the above cases of encephalitis, myelinoclasts is not a characteristic symptom. The acute demyelinating lesions found in Australian X disease, equine encephalomyelitis, and dog distemper, have nothing in common with those of acute myelinoclasts.

The following theories have been suggested to account for myelinoclasts:—

- (I) The lesions are produced by the virus responsible for the visceral lesions or by a neurotropic variant of this virus.
- (II) The lesions are produced by a demyelinating virus, activated either by other virus infections or by non-specific causes.
- (III) The lesions are allergic in character.
- (IV) The lesions are due to the combination of a virus or other infective agent and a nutritional deficiency.

If acute myelinoclasts is due to the same virus as that producing the systemic lesions, it is curious that the nervous changes in no way resemble those normally produced in the central nervous system by the viruses of variola, vaccinia, varicella, rabies, yellow fever, and influenza. Vaccinia and variola are predominantly mesodermotropic, and attack more especially the meninges (Hurst and Fairbrother,

1930), while rabies, yellow fever, varicella, and influenza produce degenerative changes in the neurones of the grey matter. The lesions normally produced in the central nervous system by these viruses are, in fact, quite characteristic for each virus, and in many instances are associated with specific inclusions; the characteristics of acute perivascular myelinoclasia, on the other hand, are so similar that it would be extremely difficult to be certain on histological grounds whether variola, vaccinia, measles, varicella, rubella, rabies, or yellow fever was responsible for a particular case of myelinoclasia. The specific inclusions are absent. In addition, there is pathological evidence that lesions indistinguishable from those of acute perivascular myelinoclasia have occurred after chronic gonorrhoea (Küssner and Brosin, 1886), while Wohlwill (1928) records similar changes in a patient not suffering from any known infectious disease. The demyelinating lesions of so-called distemper encephalitis have been found to occur quite independently of distemper by Perdrau and Pugh (1930) as well as in dogs known to be immune to distemper (Verlinde, 1939).

The occasional isolation of a virus from the central nervous system or cerebrospinal fluid, e.g. vaccinia virus in cases of post-vaccinal encephalitis, cannot in view of what is now known of the latency of viruses be taken as evidence that the particular virus isolated is the sole cause of the demyelinating lesions. The presence of a polymorphonuclear leucocytosis in the blood in measles encephalitis is not characteristic of a pure virus infection. Nevertheless, the virus infection may on occasions be very closely related to the presence of these degenerative lesions for a number of observers have found perivascular demyelination in some, though not in all cases, of acute distemper associated with nervous symptoms.

The second theory of the cause of acute myelinoclasia is that a latent virus is activated by the acute infection. Thus from time to time, claims such as those of Caminopetros *et al.*, 1938, are made of the isolation of a new virus from post-vaccinal encephalitis. Verlinde (1939) also has recently obtained a neurotropic virus, distinct from that of distemper, from the brains of dogs suffering from distemper encephalitis. The majority of observers, however, have failed to isolate any virus from the central nervous system in cases of acute myelinoclasia. Despite the fact that acute myelinoclasia has increased in frequency in the last twenty years it is not a common condition and evidence for its infectivity, apart from a slight familial tendency, is entirely lacking.

The theory that the demyelinating process is due to an allergic reaction, affecting only the central nervous system, has little positive evidence in its favour. The fact that neoarsphenamine encephalitis usually occurs after a second injection of neoarsphenamine has, however, been taken as possible evidence of a sensitization.

Certain unpublished observations carried out in conjunction with my colleague, Major R. D. Mackenzie, R.A.M.C., suggest that mice given a low-grade immunity to Rift Valley fever become more susceptible to the intraperitoneal inoculation of a neurotropic strain of Rift Valley fever virus. Increased sensitivity of the nervous system after some degree of immunization may possibly explain the findings recorded by Perdrau (1938) where herpes simplex virus after a long incubation period localized in the brain and produced encephalitis in immune rabbits. A more probable explanation is that, as emphasized by Lépine (1939) in the case of rabies, the central nervous system is less easily immunized than the other tissues of the body.

A fourth possibility in the aetiology of acute myelinoclasia is the presence of a relative deficiency in some nutritional factor, the deficiency being rendered more severe by the superadded action of a virus or other infective agent. One example of the way in which a nutritional deficiency and a virus might combine in the production of myelinoclasia may be given. Dogs fed on a vegetarian diet are, as shown by Minot (1927), more susceptible to guanidine poisoning, owing to the fact that the calcium present in the diet is insufficient to neutralize the guanidine. Guanidine, or guanidine-like substances, are produced when the liver is damaged by

chemicals such as carbon tetrachloride or viruses such as yellow fever. Guanidine, however, has been used experimentally to produce demyelination (Rosenthal, 1913) and evidence has recently been brought forward by Verlinde (1939) that guanidine given in sublethal doses to dogs predisposes to encephalitis with demyelinating lesions, if, at the same time, viruses such as vaccinia or dog distemper are instilled intranasally. These observations are in conformity with those made by Fuchs (1921) who claimed to have produced in cats an encephalitis, resembling encephalitis lethargica, by the intracerebral injection of guanidine hydrochloride, while Perdrau (1938) believes that he activated a latent infection due to Borna disease by subcutaneous injections of guanidine in rabbits. Further observations are required to determine the effects of the combination of a virus infection and deficient diets known to produce demyelination.

#### PSYCHOPATHOLOGICAL DISEASES IN ANIMALS

Psychological disorders are more frequent and far better known in man than in animals. Very little is yet known in regard to the causation of the condition known as hysteria in dogs. The disease has become more frequent in the last twenty years and although it is rare among mongrels it attacks almost all breeds of dog. An examination of the brain shows no specific changes, nor have my own attempts to transmit the disease by intracerebral injection of filtered nasal washings or brain suspensions given rise to any positive results either in dogs or other laboratory animals. The puppies used for intracerebral inoculation were, however, mongrels, and not of the same breeds as the hysterical dogs. Clinically there is, as a rule, no rise of temperature during the hysterical attacks, while in the intervals between attacks the dogs are apparently normal.

Various theories have been suggested as to its causation. The evidence for a virus or other infective agent is slight; hyperinsulism, vitamin B<sub>1</sub> deficiency, or the action of phytotoxins from a too-exclusively vegetarian diet, have been suggested as possible causes, but experimental evidence is lacking. Another disease of psychopathological interest in dogs is gun-shyness. The symptoms are quite distinct from those of true hysteria there being, for instance, no loss of sphincter control. During the last war when serving in Belgium in 1914, I had a young Berger de Grunendael, a breed of dog much used in police work by the Belgians. This animal was quite normal until on one occasion it was exposed to a heavy bombardment. Thereafter, uninjured physically, it developed symptoms of extreme terror whenever it heard any loud noise, a condition not unlike the so-called shell-shock in man.

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**Dr. J. R. M. Innes, Institute of Animal Pathology, Cambridge University:** Dr. Findlay has dealt with a large group of conditions of the nervous system common to man and animals; there are, however, many others, and it is my intention to deal with only a few examples.

#### MALFORMATIONS OF THE NERVOUS SYSTEM.

Probably more has been written about malformations of the nervous system in animals than any other condition (*see* Dobberstein, 1936; and Ernst, 1909). Most of the malformations (lethal and sublethal) occurring in man have been described, for example anencephaly, acrania, total and partial cranioschisis, exencephalia, amyelia, encephalocele, meningocele, spina bifida, macro- and micro-encephaly, various hypoplasias and aplasias, micro- and macro-gyria, anophthalmia and microphthalmia; one which has not been recorded is tuberosa sclerosis. Such studies as these are of importance from the aspect of comparative embryology.

#### PIGMENTATIONS.

**Melanosis.**—It has been stated that melanin pigmentation of the cells of the *substantia nigra* is specific for man, but Scherer (1939) has shown that it occurs in adult anthropoids, though invisible macroscopically. In some animals (cows and sheep) melanin pigmentation in association with the central nervous system is found with a frequency and degree not found in man or the apes. In these animals pigmentation of the dura and/or pia mater is a constant finding, and this has been confirmed by the personal examination of many sheep brains. In the sheep, intense pigmentation may be confined to the pia mater covering the superior frontal gyri of the cerebrum (motor area), while scattered fainter deposits may extend over the peduncles, pons, and medulla. In cattle similar changes may occur in the cerebral meninges and the spinal pia, but the latter is usually regarded as an accompaniment of generalized melanosis, affecting for example the lungs, endocardium, aorta, and liver. Even when intense, it causes no apparent functional derangement. The cause is unknown and can be regarded more as an abnormal increase of chromatophores constantly present in the meninges rather than as a heterotopic phenomenon. As the melanomatous meningioma enters into all type classifications of the meningioma in man, this condition in animals is important from that aspect.

**Lipofuscin.**—Many normal nerve cells contain lipid in the form of granules of lipofuscin and in man its presence is regarded as a normal phenomenon of unknown significance associated with advancing age. The same type of cellular inclusions has been observed in senile horses by Kikuchi (1928) and others.

**Iron pigmentation—Pallidal siderosis.**—Hurst (1934) has shown that in horses iron, in association with calcium, is often found in the vessels of the *globus pallidus*, while Kikuchi (1928) and Holz (1936) have recorded similar lesions ("pseudocalcification") in the *nucleus dentatus*. The histological appearances were comparable with the human condition, the deposits lying either in the media or adventitia, or both; in Hurst's cases the horses did not show signs of generalized vascular disease, nor could the siderosis be related to the pathological condition responsible for death. Similar appearances were seen in monkeys but not in guinea-pigs, rabbits, mice, or rats, while personal examination of the brains of old dogs has not revealed similar lesions. Hurst concluded that the changes represented a phenomenon of some constancy in higher animals of advancing age.

#### ATROPHY.

**Senile atrophy.**—Although some senile changes are well known, the effects of age on the nervous system in animals have not been studied extensively, largely because few animals are allowed to live to an age comparable to the senile state of old people. Available information (*see* Dobberstein, 1936) indicates that the changes described in man also occur in animals. The brain may be shrunken and less in weight, although

this is difficult to assess for there are no data of normal weight standards for animals of different ages; the meninges may be thickened, the convolutions separated by markedly deep and wide sulci, and the brain tissue tough. Specific human senile or presenile entities, such as Alzheimer's and Pick's disease, and conditions such as paralysis agitans and idiopathic tremor, have not been observed in animals.

#### PACHYMEINGITIS SPINALIS OSSIFICANS.

This is a pathological condition which might also come within the range of senile phenomena, and has been described by Kitt, Dexler, Joest, and others (see Dobberstein, 1936). In the cervical and lumbar regions particularly, but occasionally in the cranium, the dura mater shows bluish-white plaques which may fuse and reduce the dura to a rigid tube. These plaques are true lamellar bone with Haversian systems, and do not arise from the vertebræ for a normal epidural space is present. The bone lies in the middle of the dura thickness and is covered on both sides by connective tissue; the aetiology and pathogenesis of this condition is still uncertain. There is not sufficient evidence to show whether it is an ossification of an old-standing chronic pachymeningitis or neoplastic in nature. The effect of this dural thickening may, in severe cases, cause a compression myelitis and chronic neuritis of the nerve roots which may in their turn cause paralytic symptoms, protracted muscular spasms, and painful paresthesias. The closest parallel in the human being seems to be the occurrence of calcified plaques in the arachnoid of old people.

#### INTRACRANIAL TUMOURS.

*Gliomata* are regarded as rarities in domesticated animals, but until some thousands of animal brains are examined, no dogmatic statement can be made. The few records in the literature were published before the work of Bailey and Cushing (1926), and refer to the tumour simply as a "gliosarcoma". This only indicates that with hamatoxylin-eosin sections the tumour was highly cellular with an appearance like a sarcoma in non-cerebral sites. The case recorded by Pallaske (1935) in a dog was an astroblastoma and no histological descriptions are given of ten cerebral tumours in dogs recorded by Milks and Olafson (1936), although the diagnoses included oligodendroglioma, "ganglio-neuroma", medulloblastoma, choroidal papilloma, and "perithelial sarcomas". A few cases of "glioma" have been described in horses and these were classified as "gliosarcoma". There are no records of the occurrence of other tumours of the glioma group such as neuro-epithelioma, *spongioblastoma multiforme* and astrocytoma; an ependymoma of the lateral ventricles of a mouse has been described by Slye, Holmes and Wells (1931).

Similarly the meningioma as such has not been recorded in animals, although several tumour entities have been described as spindle-cell or round-cell sarcoma which might well have been meningioma. The "cholesteatomata" (see Holz, 1935; Dobberstein, 1936; and Critchley and Ferguson, 1928) which occur in the horse either as a dural "epidermoid cholesteatoma" or as "plexus cholesteatoma", and which have often been termed by veterinarians as "psammoma", is not a meningioma. The "epidermoid" type, which may compress the brain, contains epidermoid derivatives and has been suggested to be teratoid in nature, while the "plexus cholesteatoma" has been regarded, mainly by French and German authors, as a chronic granulomatous lesion of the choroid plexus with massive deposition of cholesterol crystals. A suprasellar craniopharyngioma in a dog has been described by White (1938) and he refers to the few records of pituitary tumours in animals.

The incidence of metastatic tumours of the brain in animals is also not known but is probably not so common as in man. (Willis (1934) gives the incidence in man as about 5% of all fatal cases of malignant disease.) I have seen in the brains of dogs, metastases of a malignant melanoma of the skin and of a primary carcinoma of the mammary gland.

## CANINE ENCEPHALITIS.

Although a so-called nervous form of canine distemper exists clinically it is now generally held that the distemper virus itself is not associated with cerebral manifestations. Infection with the distemper virus may, however, sometimes precede these nervous complications. In 1930 Perdrau and Pugh observed demyelination of the type found in subacute disseminated sclerosis in 4 out of 14 cases of disseminated encephalitis in dogs; other observations included mononuclear infiltration of the meninges and perivascular "cuffing", but there was no uniform distribution of the lesions except for the meningitis of the olfactory lobes; in most cases lesions predominated in the pons, medulla, peduncles, and cerebellum. They concluded that "distemper" was not an essential antecedent of the disease, as it occurred in only 7 of their 14 cases, but the virus played a rôle similar to that which some acute infections play in certain post-infective demyelinating diseases of man. Other papers on the same subject include those by Gallego (1928), Posrednik (1930), De Monbreun (1937), Marinesco *et al.* (1933), Peters and Yamagiwa (1935), and King (1939).

The terms "distemper" or "post-distemper encephalitis" have been loosely applied and with no more proof than that the dog may, some time previously, have suffered from a febrile illness. Scherer and Collet (1939) have recently remarked on this and described pathological entities in the brains of each of three dogs which were clearly distinct from each other; all three processes described by them have at various times been illustrated as part of "post-distemper encephalitis".

Nearly 50 dogs with a "nervous disorder" have been examined personally, and the clinical diagnosis included "fits", "hysteria", "chorea", "epilepsy", and "distemper encephalitis". Some may have been a true "hysteria" or psychopathic state as they showed no obvious brain lesions. The remainder showed varying changes indicative of the disseminated encephalomyelitis with demyelination, described by Perdrau and Pugh (also described in one of Scherer's cases), and by King (1939), while others corresponded to the process of diffuse intracortical vascular proliferation without inflammatory infiltrations and occasional foci of meningitis described in Scherer's first case. (The latter type of lesion is also described by Peters and Yamagiwa in their "distemper encephalitis" and who compare it with Wernicke's encephalopathy.) Whether yet others correspond exactly to the disseminated focal type of sclerotic lesions described in Scherer's second case is uncertain. There is, however, enough pathological evidence to show that more than one entity may occur in the dog which may have been regarded in the past as nervous forms of distemper or as "post-distemper encephalitis", and in which the association of the distemper virus is problematical.

Dunkin and Laidlaw (1926) had been unable to demonstrate lesions similar to those described by Perdrau and Pugh in their experimental cases of distemper which showed nervous manifestations. The brains of dogs with natural distemper infection examined personally have also shown no similar lesions. With Professor Dalling, attempts have been made to isolate distemper virus from the blood, brain, or spleen of cases of "encephalitis" and the results were negative. Inoculations of the distemper virus into the brain of dogs have failed to produce any distinct encephalitis, while attempts to transmit the encephalitis to other dogs have been equally unsuccessful (Dalling, personal communication).

The clinical evidence is also puzzling; in many cases there is a history of a febrile illness ("distemper"?) at periods varying from a week to more than a year before; in some the illness was intermittent. Occasionally, after the initial illness, there was a slowly progressive paraplegia with ultimate blindness. Some showed fits and convulsions passing into a flaccid paralysis, some a spastic gait, others a loss of sensation in the extremities.

The uniform time interval which occurs in man between the onset of some acute exanthematous infections and the onset of nervous symptoms has no parallel in the dog. The demyelination encephalitis in dogs is therefore not strictly analogous to the post-infective demyelinating diseases of man. As Perdrau pointed out, the variable and lengthy time interval in some canine cases is more comparable to the relation which has been stated to exist between disseminated sclerosis and a previous acute infection, and one case of Scherer's seems to bear a closer resemblance to disseminated sclerosis than the acute disease.

There is more support for the view that there is no direct relation between the distemper virus and the "encephalitis" from the failure to find the inclusion bodies which are said to be typical of distemper.

The pathology of the condition indicates that its aetiology is likely to be linked to a virus, or viruses, not yet identified. Green (*see* Green and Shillinger, 1934) states that fox encephalitis is transmissible to dogs, and that inclusion bodies are found in the ependymal and endothelial cells of the brain and in the liver. These specific inclusion bodies are stated not to be found in natural or experimental canine distemper. An experiment by Dalling and myself supports the contention of Green that the two viruses (fox encephalitis and distemper) are distinct. The former produces meningo-encephalitis in dogs in contrast with the distemper virus; in experimental fox encephalitis infection in dogs, specific intranuclear inclusion bodies may be found, particularly in the hepatic cells. A search for these inclusion bodies in the liver and other tissues of natural "canine encephalitis" cases was negative.

The disease in the dog occurs very frequently, and probably more often than the post-infective encephalitis in man. The nervous phenomena in the dog, known as "fits", "hysteria", "chorea", "epileptiform convulsions", are vague in meaning; the study of these conditions would be valuable not only to clarify their significance but also to understand better the acute and chronic forms of disseminated encephalomyelitis in man.

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## Section of Psychiatry

President—F. L. GOLLA, O.B.E., M.D.

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### Myokinetic Psychodiagnosis : A New Technique of Exploring the Conative Trends of Personality

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ALL the usual devices for exploring temperament [and character are either concerned merely with the physiological correlates of these psychological qualities, or are strictly related to situations in which there is a large number of possibilities of self-defence against them, i.e. to situations artificial and ingenious enough to allow an intelligent person a means of cheating the tester.

Recently, though considerable effort has been made to provide reliable methods of testing these aspects of the human mind, there is, as far as I know, no definite and simple technique that can be used in clinical psychiatry for this purpose.

To indicate the way in which further investigations could assure such a technique is the principal aim of this paper.

#### FUNDAMENTALS OF THE NEW TECHNIQUE

When I started my research I wanted to provide "an objective measure of the dominant (conative) trends of personality as expressed in the individual's basic attitudes of reaction". In other words, following the ideas of Kurt Lewin, I wanted to detect the "vectorial expression of the conative trends".

I was impressed by the results already obtained by Downey with her battery of tests and I thought that Allport and Vernon were right when they wrote: "A considerable simplification of some of the complex factors in graphology may be secured through an investigation of the drawing of figures and lines. Oddly enough this rather obvious procedure has hitherto been neglected" ("Studies in Expressive Movement", New York: MacMillan, 1933, pp. 81-82).

Owing to the unity of the living subject every mental attitude of reaction must be accompanied by a corresponding muscular attitude, in which the movements leading to the realization of the purpose contained in the mental attitude are to be facilitated and those opposite to it are to be rendered difficult. As far back as 1828 Chevreuil showed with his "pendulum" that merely the "image" of a movement is enough to start it, unconsciously. Hence it is to be presumed that, according to this, each subject must have in any given moment a particular set of movements which is more capable of being elicited than all the others. Consequently if we ask him to accomplish a given movement in all possible directions of space it may be expected that this movement will be better executed in the directions corresponding to that predominant set, and will be more or less hindered in the opposite directions. As a matter of fact we can even assume that the differences so detected can be used as significant of the habitual attitude of reaction of the individual, provided that we find them sufficiently constant.

W. Stern pointed out that there are considerable individual differences in the

subjective estimation of space according to changes of mood and purpose. Probably this can be explained by the same fact, i.e. the peculiar adjustment of the motor sphere to the potential satisfaction of the predominant conative drives in any given moment.

On the other hand, some of the well-known techniques of character analysis (such as physiognomy, graphology, chirolgy) and the expression of personal trends in the art of the film are, more or less, based on this, although none of them is reliable enough to be employed or advised for clinical purposes, too many factors being involved in the study of such elements as facial expression or handwriting.

Myokinetic psychodiagnosis, as I call it, is based on the involuntary expressions of the predominant attitude of reaction evaluated as a function of the shiftings observed during blind execution of linear movements in the fundamental directions of space.

*Material required.*—(1) A square or rectangular table and chair in which the subject may sit comfortably; (2) a wooden board, 15 in. long and 12 in. wide; (3) three sharp pencils; (4) one box of drawing pins; (5) six sheets of white paper 12½ in. long and 8 in. wide; (6) a handkerchief or any rectangular shape that may serve as screen (to be interposed between the paper and the subject's eyes in order to deprive him of the visual control of his hands); (7) a metric ruler.

*Instructions.*—The subject is instructed to sit down comfortably in front of the table, in such a way that his nose points straight at the centre of the paper on which he is going to work. A sheet of paper has been previously attached to the wooden board by four drawing pins, one on each corner (care must be taken that these pins cannot catch the forearm of the subject).

A preliminary and important measure is to be sure that the edges of the board are parallel with those of the table. The subject is told that he is not to be allowed to change the relative positions of his body, the chair, and the board, either before or during the execution of the test. (This is necessary because most people have a habit of bending when intending to draw or to write.)

Once the correct geometrical position of the elements involved in the execution of the test has been obtained and checked, the experimenter says:—

"I am going to ask you to draw some lines in order to detect the accuracy of your movements. Hold this pencil with your right hand and draw ten lines like this one (he draws a horizontal line 5 cm. long, going from left to right with his pencil). Before you start, I must tell you that you are to try to draw all of them the same length, parallel, and as close as possible to one another, starting and ending on the same level, like this (the experimenter draws two lines, one beneath the other, below the model, trying to keep them as equal as possible). You are not allowed to draw these lines as if you were writing, resting your wrist on the table. Your hand must be free and should be moved loosely, with its movement directed by the forearm just as I am showing you now (the tester imitates the movement he is asking for). You can, if you want to, move your hand along the paper when drawing, provided you do not fix it and you do not attempt to make movements with your fingers like this (the experimenter shows the wrong way of doing it)".

Before the subject starts, the tester continues: "Of course, it would be very easy for you to do just as I have told you, but in order to increase the difficulty you are not allowed to see what you are doing. So I will blindfold you with this handkerchief (or, interpose this screen between your eyes and your hand) and you have to keep on drawing merely guided by your feelings and without any possibility of visual control." Once this has been done the tester must guide the hand of the subject to the proper point where he should start to draw. The tester repeats: "Remember you will draw the lines from left to right, one beneath the other, and as close, equal, and parallel as possible."

The experimenter must watch the subject carefully during the execution of the task in order to avoid any omission in the observance of the instructions. He must, whenever possible, take the time needed for this performance, and if the subject mixes the lines he will try to recall the order in which these lines have been drawn in order to be able to indicate the proper order on the paper when the task is finished.

After having drawn an arrow to indicate the direction of the lines that have just been made by the subject (eventually the initial of the drawing hand is added) the tester goes on with the instruction: "Draw ten lines more, under the same conditions but going in the opposite direction, i.e. from right to left." (The previous precautions being again observed).

"Now take the pencil with your left hand and try to repeat what you have done with your right hand. That is, you must draw ten lines of the same size as the model, all equal, parallel, starting and ending at the same level and as close as possible to one another." "You must start drawing now from right to left because it is easier." (Once the lines have been drawn and the corresponding indications have been made the subject draws another ten lines with his left hand, in the opposite direction, i.e. going horizontally from left to right.)

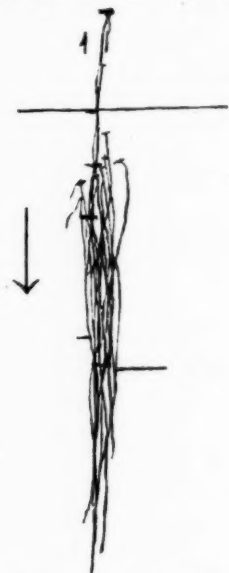
Having collected the 40 horizontals on the lower half of the paper the tester turns the board round and proceeds to obtain the lines on the sagittal plane as follows: "Now I am asking you to draw with your right hand ten lines more, going outwards, like this (the tester draws a line going on the suggested direction on the right side of the paper); this line, 2 in. in length, must be drawn quite straight and perpendicular to the lower edge of the paper. (It is worth mentioning that the average subject interprets this direction as being upwards, although it is really outwards from him.) In order to get them as close together as possible you will try to draw all of them on top of the first, i.e. on the same spot. Remember that all must be equal, in such a way that after being made they should appear as a single line. Be careful to keep your hand moving freely and loosely all the time."

The tester must carefully note the sequence of these lines. Whenever possible special signs are to be put beside the lines, Nos. 1, 2, 9, and 10, which are more significant from the statistical point of view. Once the observations and registrations have been made the subject is asked to draw more lines in the opposite direction (*inwards*, that appears to be *downwards* to him) observing the same rules. Immediately after, the corresponding records of the left hand are to be taken. It must be assured that the drawing with the right hand takes place in the right half of the paper, the one of the left, in the left side (otherwise a factor of comfort or discomfort would interfere with the results).

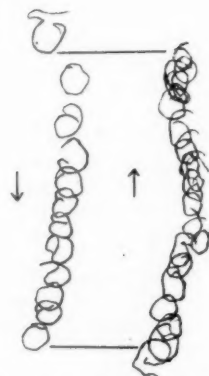
A new sheet of paper having been fixed and prepared, the board is placed vertically and the tester proceeds to obtain the corresponding 40 lines in this plane, as follows: "Now hold the board with your left hand and try to draw ten lines going up like I do (the tester draws the model) all equal, and on top of the first. Remember your hand must move along quite loosely, without resting on the paper; try to keep all the lines on the same level and of the same length." Special care must be paid to prevent the subject resting his elbow somewhere; all the verticals must be drawn with the hand and the arm in the air. In order to avoid possible oscillation of the board the tester must also hold it with his free hand (the other hand holding the screen if the subject has not been blindfolded). Once the ten verticals going up have been obtained the tester asks the subject to draw the ten verticals going down and afterwards he gets the corresponding verticals of the left hand.

120 lines have now been obtained, 40 on each of the three fundamental planes of space. Of course, if any abnormality is observed in the collection of these lines it is necessary to repeat the trial until the corresponding lines have been correctly performed. With this the first part of the test comes to an end. There is then a rest of five minutes and the second part can follow immediately.

The second part of the myokinetic psychodiagnosis consists in the drawing of some simple combinations of straight and circular lines in the sagittal and the vertical plane in order to: (a) Check the results obtained in the first part; (2) learn something of the peculiarities of conation that are to be observed during the execution

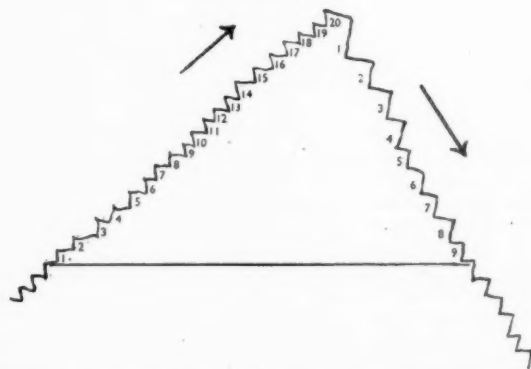


Falling of the verticals. Right hand. Scale  $\frac{7}{10}$ .



Chain test. Left hand. Scale  $\frac{1}{2}$ .

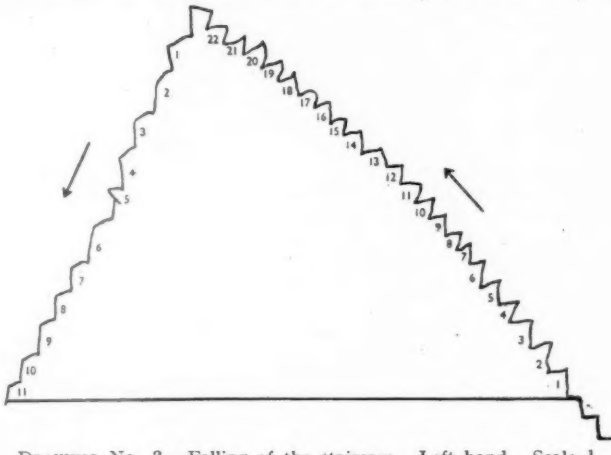
DRAWING NO. 1.



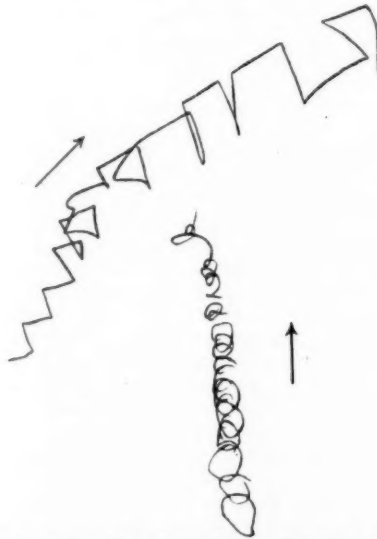
DRAWING NO. 2.—Falling of the staircase. Right hand. Scale  $\frac{1}{2}$ .

of combined movements (specially of those linked to the "shape" of these movements). The following description is an abbreviated form of this part of the technique. It implies the use of the "zigzag lines" and the "chain" tests in the sagittal plane and the "chain", the "staircase" and the "top of the castle" tests in the vertical

plane. Starting with the first, here is how we proceed: The subject is provided with a well-sharpened pencil in each hand and requested to continue two zigzag lines which the tester has drawn on each side of the upper edge of the paper. The



DRAWING No. 3.—Falling of the staircase. Left hand. Scale  $\frac{1}{2}$ .



DRAWING No. 4.—Reversion of movements and loss of direction during the staircase test in a schizophrenic patient. Note clumsiness of circle drawing. Scale  $\frac{1}{2}$ .

board is put as usual well centred, and the screen is interposed in order to keep the performance out of the visual control of the subject. But he is allowed to see where he starts and it is sometimes convenient to let him draw the first two or three

lines, before the screen comes into action. Since the zigzag lines are on top of the paper, he is drawing *inwards*, although he usually thinks that he is drawing *downwards*. A very important point to be watched is that the movements of both hands must be carried out simultaneously and not successively. In order to obtain this it is convenient that the tester holds the subject's wrists and shows him the sequence of movements to be done (which corresponds to a separation and a close-in of his hands). The model lines are 1 in. in length and with an angle of separation of  $10^\circ$ . The subject is specially requested to keep all the lines equal and to continue his drawing until the tester tells him to stop. Once he has reached the half of the sheet, the tester stops him, reverses the board, draws two zigzag lines more, going now in the opposite direction (i.e. *outwards* or, according to the subject's view, *upwards*) and asks the subject to follow the drawing towards the top of the paper.

In the reverse of the same sheet of paper the "chain" test can be done in this way: The experimenter draws four circles of about  $\frac{3}{10}$  in., each intersecting in a chain form, in the lower part of the right-hand side of the paper, and says to the subject: "Try to draw a chain like the one I have started. You will first draw it going towards the top of the paper and when I tell you you will stop, raise your hand, and continue to draw it in the reverse direction, i.e. towards the bottom. You must keep your hand in the air when drawing, try to make all the circles equal, and draw them straight up or down, without bending the chain." In order to get the best results it is convenient that the subject starts to draw the chain with his right hand in such a way that the arm is hanging perpendicularly to the plane of the table and the forearm and hand follow the same axis in the horizontal plane (which implies that the board will be centred according to the axis of the arm instead of being centred according to the axis of the nose as it had been hitherto). Once a deviation of  $30^\circ$  (approximately) from the initial position of the arm has been reached in the drawing, the tester asks the subject to stop and to reverse the direction of the chain until he has come back to the initial position.

The same instructions are to be observed for getting the chain drawn by the left hand.

This "chain" test must be accomplished too in the vertical plane, i.e. with the board in a vertical position. The subject is allowed to hold it with his free hand (the tester does the same). He is asked to start the chain from the middle of the sheet going up, first, towards the top, and reversing the direction when requested to do so (the tester will give this order to reverse when he sees that the subject is approaching too close to the end of the paper). The time of each performance must be registered and is almost as significant as the result of the drawing itself. Special warning must be made in order to avoid the subject stopping during the performance and restarting again (thus resulting in a "break" of the lineal shape of his movements). The chains with the left hand will be obtained immediately after those of the right hand, under the same conditions and instructions.

Another important complement of the examination is afforded by the execution of the "staircase" test, which is to be done after a few minutes' rest and consists in asking the subject (with the board and hands in the same positions as for the preceding exercise) to draw a staircase, according to a model previously drawn by the tester. Two staircases must be drawn with each hand, both of them are to be complete, i.e. must go up and down, but will start at opposite points and will be drawn in opposite directions: First, the tester starts a staircase on the left centre of the paper and draws it going to the right, with the right hand. Once he has drawn three steps he asks the subject to follow it, going up to the right until he is instructed to go down and continue to the right. The steps must be all equal and the subject is asked to ensure that the change of direction in the drawing does not affect its size or its shape. Each step will be drawn  $\frac{1}{10}$  in. long, and  $\frac{1}{10}$  in. high by the tester. Once the first staircase with the right hand has been obtained the subject is asked

to draw the corresponding one with his left hand (starting on the right centre of the paper and going up towards its left and finally going down still to the left). Immediately after the two other staircases are obtained (the one going from right to left with the right hand and the one going from left to right with the left hand).

Finally, it is convenient in the majority of cases (the minority being provided by those in which the subject is reluctant to be examined or shows clear signs of fatigue) to end this second part of the myokinetic psychodiagnosis with the "top of the castle" test. This is similar to the staircase test, with the difference that instead of a staircase the subject is invited to draw four battlements representing tops of a castle. These drawings must be made on the top of the paper, the board being vertical as in the preceding tests; first the subject starts to draw from left to right with his right hand, following to the right across the paper; then he draws from right to left with his left hand and finally he repeats the drawing going from right to left with the right hand and from left to right with the left hand. He must be specially warned to hold not only the hand but also the elbow in the air, and to try and keep the same level of height during the execution of the test. The time must be also registered.

In special cases, and in order to make clear some doubtful results, other tests can be used, such as the drawing of spirals, the drawing of circles on top of each other, and the drawing of lines followed by its crossing in the middle. This last procedure, specially useful for checking the results obtained in the sagittal and the vertical planes, allows a better understanding of the raw and sometimes contradictory results that may be obtained if only the first part of the examination is made in unstable psychopaths. This point is made clear in the clinical material.

#### EVALUATION OF THE DATA

To get information for clinical purposes it is not necessary to exhaust this analysis; and, furthermore, some psychological data cannot be derived from it which can be gathered from the drawings. The following technique of evaluation of the tests is a tentative one which has given good results, although some of its rules can be questioned from a purely mathematical point of view:—

To begin with the first part of the myokinetic psychodiagnosis, it is possible to differentiate (a) the quantitative data; (b) the qualitative data. Among the first, the following are the most convenient:—

✓(1) The length of lines; (2) the average length of lines corresponding to each hand in the different planes and all together; (3) the variability of these lengths; (4) the sense and amount of the *general* variation of length of lines corresponding to each hand; (5) the absolute and relative shiftings of each sequence of ten lines; (6) the corrected averages of these shiftings as compared with those that should occur if the subject would have been able to keep the length and the separation of lines according to the instructions; (7) the coefficient of coherence of the measures, which is to be obtained by dividing the average of the relative shiftings by the average of the absolute shiftings.

All the tests being repeated after seven days, the correlation between the relative shiftings (i.e. the corrected averages of the shiftings of each sequence of lines considered algebraically) is to be obtained and its value will be considered as a measure of the reliability of the test in that case.

Among the qualitative data (which in some respects could also be submitted to quantitative expression) we include the following: (a) the straightness of lines; (b) their load; (c) their regularity, considered from the point of view of their orientation in space and of their general appearance.

These qualitative elements are of primary importance in the second part of the M.P. where figures and shapes must be considered. In these materials it is possible

to detect, in addition to all the measures and data already mentioned, more psychological expressions of the subject : personality ; the care, meticulousness, or disregard with which the performance has been made, its gracefulness or, on the contrary, its clumsiness, and some other trends of character may be revealed in this way. It is impossible to give all the details here.

Coming back to the quantitative measures it is necessary to explain what I mean by such terms as general variation of lines, relative and absolute shifting. The first is to be obtained by adding all the differences of length of all the lines drawn with the same hand and the length of the model (if the addition is made in an arithmetical way the absolute variation will be obtained ; if it is made in an algebraical way the relative variation will appear) and by dividing the sum by the number of lines. In order to detect the sense (positive or negative) of this variation, it is better not to refer it to these differences but to the total number of lines which are longer and the total number of lines which are shorter than the model. If the first is bigger than the second the quotient of both will be superior to 1 and I call it a positive (increasing) sense of variation ; if the contrary happens (quotient inferior to 1) I call it a negative (decreasing) sense of variation, although it is obvious that in both cases we are dealing with positive figures.

For detecting the relative and the absolute shiftings which, after all, are the most significant measures of the M.P., it is necessary to determine the centres of each sequence of ten lines, to draw a perpendicular line to the first one, starting from its centre and crossing all the others of the series ; then, the distances from the centre of each one of these lines to that perpendicular is to be measured in millimetres, thus getting nine numbers which will be considered as positive or negative according to the side in which they lie ; if they are on the side of the perpendicular which corresponds to the suggested direction of movement they will be quoted as positive, and they will be considered as negative if they lie in the opposite side. If the tendency to shift is a constant one, it is to be expected that in each sequence we will find merely positive or negative values ; the mixing of positive and negative shiftings in the same lineal series points out that the tendency is being hindered by some opposite factor (conscious control or autonomic subconscious drive).

If now we add all these nine numbers arithmetically and get the average we will know the amount of the absolute shifting of this given sequence of lines. If we add them algebraically and get the average we will know the sense and the amount of the corresponding relative shifting.

Working with groups of subjects instead of working with individual cases gives quite a number of significant measures which will be explained and discussed in the interpretation of the obtained results.

#### PRELIMINARY RESULTS

I have applied the M.P. to 187 persons, of whom 38 were supposed to be mentally normal and 149 were considered as mentally unbalanced. 19 of the normals have been retested after intervals varying from three to thirty-four days. 54 of the abnormals have also been retested at approximately the same intervals ; three cases have been tested three times ; two have been tested four times. As the minimum number of lines to be worked out in each case is 120, though in many cases this number was far more (up to 350 in one observation), it is possible to estimate roughly about 24,000 lines. I am not giving the results of all for various reasons : (a) some cases are not well defined from the clinical point of view ; (b) others have not been in a condition to end the test ; (c) some of the results are still to be worked out from the statistical point of view. In all I am trying to sum up the principal facts which emerge from the results obtained in 145 cases, the majority of which have been

retested, thus allowing us to judge the reliability of their results. The distribution of these cases runs as follows :—

✓Normals .. ..	35	(18 females; 17 males)
✓Epileptics .. ..	32	(19 females; 13 males)
✓Depressed .. ..	16	(6 females; 10 males)
✓Anxiety dep. .. ..	12	(7 females; 5 males)
✓Schizophrenic .. ..	16	(14 females; 2 males)
✓Elated .. ..	7	(2 females; 5 males)
✓Psychopaths .. ..	7	(4 females; 3 males)
✓Obsessed .. ..	7	(3 females; 4 males)
✓Suicidal .. ..	8	(5 females; 3 males)
✓Organic (neur.) .. ..	5	(1 female; 4 males)

In spite of the duration of the M.P. (thirty minutes average) all our subjects, schizophrenic and elated included, have enjoyed it and we have not had serious difficulties in getting the records. Those who at the beginning seem reluctant to undertake the test are the cases of severe depression, approaching stupor or mixed with an element of anxiety; in the first the inhibition is an obstacle to be overcome by the patience of the tester, who in certain cases must help them to start; in the second, the mood is usually tinged with anger which is quickly directed against the tester in the harmless way of critical and depressing remarks, unfriendly looks, &c.; owing to their lack of decision they can also be managed if the tester combines patience with psychological skill. Working with schizophrenics it is useful to start with their left hand when they find it difficult to control the right.

Of course the general behaviour of the subject during the execution of the test provides quite a lot of interesting data to the experimenter; especially his retrospections must be quoted if spontaneously expressed.

The results can be given either in arithmetical or in geometrical form. We will try to combine both ways in order to make this presentation as clear as possible. Starting with the control group of normals, let us see which are the main results.

The sequence of the absolute and relative shiftings will be represented in the following order :—

	Horizontals		Sagittals		Vertical lines	
	L.R.	L.R.	L.R.	L.R.	L.R.	L.R.
	↙	↘	↓	↑	↓	↑
Order of the measure	1st-2nd	3rd-4th	5th-6th	7th-8th	9th-10th	11th-12th

L.L. = Length of lines (average). The letters "L." or "R." correspond to left or right hand and the signs + or - express the positive or negative sense of the subsequent data. C.C. = coef. of coherence C.R. = coef. of reliability.

Allowance must be made for the fact that the males of this group are to be considered as selected samples from the average male of mankind, thus explaining, perhaps, the general outline of their results. As for the females, they too are a selected group. Anyhow, we think that both can be taken as controls of the abnormal groups, because these are also formed by rather cultured and mentally well-equipped patients.

Table I shows the slight differences that the absolute values have in males and females, and the most significant differences of the corresponding relative values.

Thus, for instance, the A.S.a. (average of absolute shiftings) in males is 6.15 and in females 5.85, but the corresponding R.S.a. (average of relative shiftings) are: 2.2 and 0.32 respectively. Consequently, the C.C. of males is 0.35 whereas that of females 0.054.

The coefficient of reliability of this group ranks between 0.69 and 0.95 with 0.80 as average. If we now compare the reliability of the shiftings of each hand we see a striking difference, those of the left being far greater than those of the right. In fact the average of reliability coefficients of the shiftings of the left hand in 20 cases is 0.88 and the reliability of the corresponding measures with the right hand is 0.73. This difference will be increased when we compare the results in certain groups of mental patients, such as for instance the psychopaths.

It is surprising that the absolute and the relative shiftings of both hands are practically equal in the group of normal subjects, although the majority complain about their inability to control their left hands (in fact they are slightly inferior in the left hand of the males).

TABLE I.														
NORMAL														
Males—	HORIZONTALS				VERTICALS				TOTAL				Normal	
	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.		
B. . . . .	-8.5	2.8	-6.6	-12	-11.6	-9	8.5	2	-5.8	-12	-2.9	-3.6	Total average of shifting with each hand	
L. . . . .	-6.5	5.9	6.2	9	3.6	9.5	-6.9	-6.9	3	6.5	-7.8	-11.6		
L. . . . .	-3.8	-0.6	4.4	4.3	-6.5	3.9	-0.6	8.3	-6.4	9	11.6	7.6		
S. . . . .	-2	5.9	-2	3	-1	3.5	-5.1	3	0	-0.2	9.1	-6.6		
I. . . . .	-22	-9.2	8.8	6.2	1.9	14	6.9	-3.6	-2.6	5	9	10.6		
B. G. . . .	-7	10	12	-15	-7	6.5	-2	8	-7	-3.5	-3	1.5		
D. J. . . .	-9	12	2.5	-2	8	8.5	5.6	15	9	5	4.8	12		
W. C. C. .	6.7	5	-11	11	3	5	5	9	8	12	10	7		
W. W. . . .	32	4	5	-6	-1.5	0	-11	1	-5	1.3	-6	-6		
G. . . . .	0.3	3	4	-2	10	6	-3.5	3.6	-1.6	-0.2	2.8	-1		
S. . . . .	7.5	26	6	-15	9	-0.2	-4	-0.5	2.2	0	1.3	1		
S. . . . .	1.2	-2.2	-6	-21	3.6	10	-11	-12	-1.2	-16	12	-8		
E. M. . . .	-1.1	16	1	6	-6.2	-5	14	12	-10	-4	6.8	0		
J. H. . . .	-4.4	6.6	0.4	3	-3.6	2.1	-3	2.4	-1.1	-1.3	-6.2	2.7		
M. . . . .	-8	4.6	-2.8	7.9	3.8	-2.4	-1.6	-4.5	-0.8	-3.9	-2.3	-4.6		
J. . . . .	-10	7.4	-0.9	8.5	-3.1	-2.7	2.7	1.7	-1	-3.9	2	-9.2		
McC. . . .	-9.2	11.6	0	2.8	-14.8	-3	12.8	10.2	-2.2	-16	-7.2	-4		
Abs. shift.	139.2	132.8	79.6	134.7	98.2	91.3	104.2	103.7	66.9	99.8	104.8	97	L. R.	
Average	8.2	7.8	4.7	7.9	5.8	5.4	6.1	6.1	3.9	5.9	6.2	5.7		
Relat. shift.	-99	120.8	50.3	61.7	-12.6	69	1.4	76.2	-44.7	-61	69.4	-12.2		
Average	-5.8	7.1	3	3.6	-0.7	4	0.08	4.5	-2.6	-3.6	4.1	-0.7	-1.9	2
Females—														
H. . . . .	-7.8	13	11	14	4.3	5.4	9	15	-7.3	2.7	9	1.4	L. R.	
B. . . . .	-18	-15	-4	15	-8	-2	-4	-5	-2	2.8	-5.7	-5		
B. . . . .	-1.8	5.6	8	-5.5	-2.5	0.4	7.8	8.5	5	-0.7	-2.4	-0.9		
R. . . . .	-0.6	8.2	-15	19	-2	-0.1	-4	-3	-4	-3.2	6.4	-2.7		
S. . . . .	-3.9	-15	-6.4	6	5.1	-3.1	-3.6	5.6	15.3	4.3	-14.3	-0.7		
M. M. . . .	-27	-2.1	1.5	4.2	1.7	-9	18	1.1	-2.2	-4.1	8.9	-1.3		
B. . . . .	16.6	6.4	-3	-12	5.8	-1.5	-3.9	-4.8	-7.3	1.3	13	-4.6		
J. V. . . .	8.6	3	6	-5.4	-4	-1.4	-1.5	-0.4	0.7	0	-0.7	-3.9		
F. B. . . .	6	-1.7	6	7.6	-4	-3.6	1.6	1.7	-6	-10	0.3	8.9		
P. A. . . .	15	-5.2	9	5.3	-12	4.3	-3	-2.5	-8	-5	3.5	10		
M. G. B. . .	-0.6	-6.3	-7	-5	-9.5	-8	-2.5	-12	1.8	9	-4	-5		
H. W. . . .	-15	7.4	2.5	1.5	14	-2	-8	-12	5	-13	8	-18		
D. H. . . .	8.1	5.5	0.8	3.9	-2	4	10	5	-10	2	-2	-9		
F. . . . .	-11	-6	1.3	15	-3.4	-6	-17	0.2	8	14	-4	-7		
W. . . . .		6.8		9.9	-4.4	2.2	5	-4	4.7	-2	1.9	3.2		
Y. A. . . .	0.9	8.2	-5.9	-0.6	-16	-6.9	7.2	-0.1	-0.2	-5.8	5.6	17		
C. . . . .	-6.5	7.9	-6	0.2	1.9	-0.6	4.8	-0.9	4.6	5.6	10.8	-5		
G. . . . .	-14	5.6	-4	-1.6	3.6	-2	-3	5.7	2.8	-3	-5.3	1.8		
Abs. shift.	161.4	128.9	97.4	130.8	104.2	62.5	113.9	87.5	94.2	88.5	104.8	105.4	L. R.	
Average	9	7.1	5.4	7.2	5.8	3.5	6.3	4.8	5.2	5	5.8	5.8		
Relat. shift.	-51	26.3	-5.2	71.5	-31.4	-29.9	12.9	-21.9	0.9	-5.1	29	-20.8		
Average	-2.08	1.4	-0.29	4	-2.7	-1.6	0.71	-11.2	0.05	-0.28	1.6	-1.1	-0.45	0
Average length of lines														
Males						L.		R.						
Females						48.3		43.6						
						45.1		40.43						

If we now compare the difference between the absolute and relative shiftings we will see that what we could call the coherence of the group is relatively small in the males (measures 0.35) and very small in the females (0.054), or in other words that we found in it a wide mixture of personalities. The compensation of the opposite

conative trends of such personalities is responsible for the low values of the averages of the relative shiftings. Another fact that we must consider as being very important is that the reliability of a given hand in two different testings is greater than that of both hands in each set of tests. This means that each hand is shifting and behaving under different trends, those of the right being more inconstant than those of the left (if we take into account the greater variability of its measures).

### Epileptics

The general results of this group are shown in Table II.

TABLE II.—EPILEPTICS.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.
<i>Males—</i>												
L. C. R. . .	-12.6	-1.8	7.4	3.5	12.6	3.8	-15.6	-18.5	-9	18.5	16	15
P. G. . .	-12.6	29	-3	4.1	19	0	3.5	12	23	5.8	16	14
J. A. H. . .	-0.3	10.3	23.6	21	5.6	12.2	-10.9	-11.6	18	8.7	-3	-3
O. . .	-3.6	13.1	-7.4	-3.4	-2.5	-4.8	-0.8	14.2	3	-3	-2	4
B. . .	-25	3.6	11	13.5	10	10	8.9	14.5	16	4.5	-20	8
C. . .	6.6	6.5	3	14.6	10.5	19	-18	15	16	20	-12	-14
N. B. . .	-3.6	9	10.6	-10.5	8.3	3.4	-4.5	-6.4	5	13.5	-7	-9
J. S. . .	12	13	8	-4.5	-5	6	13	10	15	15	-10	-12
W. Y. . .	10	-7	-2.5	7	9.5	-3	17.4	10.5	4.5	17	0.5	-12
F. A. . .	-31	3.6	14.1	-6	6	10	-4	3.8	-9	10	11	23
M. C. . .	-5.6	-3.8	1.8	14.5	-8	6	-7	-5	20	-3	-11	3
J. L. . .	-4	21	18	-3	10	10	9	9.6	9.5	20	16	12
H. . .	0	2.9	28	-4.9	-4.7	9	4	1.2	8	4	14	7
Abs. shift.	126.9	124.6	138.4	110.5	111.7	97.2	116.6	132.3	156	143	138.5	136
Average . .	9.8	9.6	10.6	8.5	8.6	7.5	9	10.2	12	11.0	10.6	10.4
Relat. shift.	-69.7	99.4	112.5	45.9	71.3	81.6	-5	19.3	120	131	85	36
Average . .	-5.4	7.64	8.65	3.5	5.5	6.3	-0.4	1.5	9.2	10	6.5	2.7
<i>Females—</i>												
J. B. . .	1.3	-6.3	-1.6	26.5	-9	5.5	12	4	18	-4	14	30
L. C. . .	-2.6	-3.9	24	0.6	-6	-2.2	15.8	9.8	14	7.8	20	-6
R. R. . .	-9.6	19.5	21.7	23.1	-13.7	3.3	11.3	12.1	-14	-2	24	11
G. . .	-12.7	-10.3	0.5	15.2	1	-6.4	-16.2	-6.6	8	-4.5	10	-3
P. . .	-4.1	-4.6	7.2	8	9	15.3	9	6.8	2	-5	10	-8
R. . .	-1.9	12.5	15.4	1.4	6	6.5	-4	8	19	5	8	2
D. B. . .	1.5	25	14	16	13.5	8.9	17.8	5	-8	-4.8	23	2
M. B. . .	-14.5	-12.5	21.5	10	0	4.1	3.2	7.8	-2	6.8	3	4
M. L. . .	7	9.8	2	10.8	2.5	6.9	-1.9	11.5	-2	8.5	2	-4
R. W. . .	-10.8	4	-19	-12	4.2	-6	-6.7	18.5	-2	3	-7	1
E. R. . .	5.6	1.2	-6.5	6.5	6.6	-5.8	-8.5	-6	8	-10.5	-9	3
E. M. . .	-5	-2.3	4	-11.4	2	1.8	-0.5	-2.5	-13	7.2	7	6
N. S. . .	11.4	7.9	20	19.3	-7.9	-8.9	-5.6	6.8	3.5	-4.8	-6.4	-7
M. G. . .	-5	-5.2	-0.8	3.9	8.6	-8.5	-3	24	11	17	2	-9
D. F. . .	-1.7	-1.8	10.5	-3	-6.5	-2.9	9	4	-11.6	-18	13.6	8
F. W. . .	-9.6	12.1	8.5	9	1.8	-2.5	9.8	6	-2	-0.6	19.6	16
E. H. . .	-4.7	10.1	16	-3.8	-10	-6.9	-9.5	4.2	-5	-5	-11	-16
R. B. . .	-3.9	7	15	-3.5	-6	-6	-8	1.8	4	2.3	9	11
Et. H. . .	-10	-1.6	10.3	27.8	3.8	-20	13.4	18	-2	-8	-5	-1
Abs. shift.	122.9	157.6	218.5	211.8	118.1	128.4	165.2	163.4	149.1	124.8	203.6	148
Average . .	6.5	8.3	11.5	11.1	6.2	6.75	8.7	8.6	7.8	6.5	10.7	7.8
Relat. shift.	-69.3	60.6	162.7	144.4	-0.1	-23.8	37.4	138.2	25.9	-9.6	126.8	40
Average . .	-3.6	3.2	8.6	7.6	-0.005	-1.25	2	7.3	1.4	-0.5	6.7	2.1

If we compare the reliability of the measures in each hand we find it still greater in the left (0.84) than in the right (0.76). It is to be noticed that the difference in the left hand values is especially remarkable in the horizontal lines, while those of the right hand values are more noticeable in the vertical lines (probably owing to the effect of the luminal treatment).

There is a clear tendency to a positive variation of the length of lines in this group, especially in the vertical plane, as the following table shows:—

AVERAGE LENGTH IN MILLIMETRES OF THE 2ND AND 9TH LINES  
IN THE VERTICAL PLANE IN 32 EPILEPTICS.

		Lines going up		Lines going down	
		Left	Right	Left	Right
Females	..	39-58	47-56	50-57	45-51
Males	..	45-60	43-52	43.5-55	45.6-53

The coherence of this group is almost three times as large as the one of the normal group (C.C. = 0.45). Difference in the load of the lines is remarkable in certain cases, those of the left hand showing sometimes a normal strength. The most important fact shown by this observation and for which I have no explanation to advance is the failure of the majority of patients to draw the staircase from right to left (especially with the left hand).

#### Obsessed

The reduced number of observations belonging to this group is not sufficient to allow us to come to any definite conclusion. Generally speaking, their results correspond to what may be expected beforehand and show a great deal of coaction in the right hand movements. The tendency of the left hand to overlap the lines in almost all directions seems to be very clear as is, too, the accuracy of the right hand in drawing geometrical shapes. The shifting averages are shown in Table III.

TABLE III.—SEX-OBSSESSED PATIENTS.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.
	↙	↘	↗	↖	↓	↑	↗	↖	↓	↑	↗	↖
	Horizontals				Sagittals				Verticals			
W. E. D.	2.4	-15	6.4	5.5	13	-35.5	25	9.5	-2.6	-5	-5.9	3.3
D. . .	-1.9	15.4	50	19.9	25	4.5	-8.4	13.8	3.3	6.3	18.4	-2.9
T. . .	-8	-12.5	6	9.5	6	-2.1	8.5	12	3.2	-6	9	12.5
B. . .	-4.9	-4.4	13.3	21.4	0.9	5.1	5.3	-9.1	9.2	8	3	-8
M. . .	-12.3	9.6	31.9	9.6	16	10.8	-1.5	9.4	-20	16	0.6	-5.2
D. . .	2.5	-1.5	25.8	13.6	10	13.8	1.6	14	12	-5	-3	-16
Abs. shift.	32	58.4	133.4	79.5	70.9	61.8	50.3	67.8	50.3	46.3	39.9	47.9
Average . .	5.3	9.7	22.2	13.2	11.8	10.3	8.3	11.3	8.4	7.7	6.66	8
Relat. shift.	-24.2	-8.4	133.4	79.5	70.9	-12.4	29.5	49.6	5.1	14.3	22.1	-16.3
Average . .	0.6	-1.4	22.2	13.2	11.8	-2.1	4.9	8.3	0.8	2.4	3.7	-2.7
			L.	R.					L.	R.		
Total av. abs. shift. . .			10.44	10.03	Av. length of lines		=		63.2	44.3		
Total av. relat. shift. . .			8.22	5	C.C. = 0.64							
	C.R. = 0.87 (extreme values 0.82-0.95)											

#### Depressed and Anxious

This is a rather heterogeneous group of cases whose clinical diagnoses are different, but in all of whom symptoms of depression and anxiety were present at the moment of the testing, thus allowing the observation of rather conflicting tendencies in each individual. All the relative shiftings are positive except the one of the left hand going horizontally towards the left, which has anyway the slightest negative value of all the groups. I think this fact expresses what we could call the "emotional incontinence" of these cases. The coefficient of coherence of this group is 0.49, which is not sufficiently high for classifying it as satisfactory. Extremely important is the positive variation of lines in the vertical planes which is larger than that found in the epileptic group. In order to detect the influence of anxiety we may compare the results of this group with those of the group of depressed patients with retardation and without anxiety. The results are shown in Table IV.



*Depressed with Retardation*

The absolute and the relative shiftings of this group are far more coincident than those of all the preceding groups, thus giving a high coefficient of coherence (0.86) and defining it as a homogeneous group. If we consider now the direction of the shiftings we will see that there is a definite tendency of all the group to give highly positive shiftings in the lines going down on the vertical plane and highly negative shiftings in the lines going up on the same plane, or in other words, that the lines drawn by these patients show very clearly the attitude of reaction in which they found themselves (Table IV A, Drawings Nos. 1, 2 and 3).

We notice in all these drawings the difficulty with which the patients accomplish the ascendant movements and the ease with which they do the descendant movements. Not only the accuracy but also the speed of the descendant movements is greater, thus resulting in a striking difference in the corresponding drawings. The most important fact is that the intensity of such differences closely corresponds to the intensity of the depression as considered from the clinical point of view, thus enabling us to get a very high correlation between the ranking of the patients according to these two points of view. A clear proof of this lies in the fact that the averages of shiftings in the vertical lines of a group of nine suicidal patients are definitely bigger than those of the non-suicidal, as can be seen in Table V.

TABLE V.—EIGHT SUICIDAL PATIENTS.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.
	↖	↖	↗	↗	↓	↓	↑	↑	↓	↓	↑	↑
T. . . . .	22	12.2	22	38	39	14.4	7.7	14	56.7	6.2	17	-4.3
M. . . . .	0	13	8.5	4.2	36	26.4	6	15	50.3	3.6	26	33
B. . . . .	-8	4.2	30	18.9	9	13.5	5.6	8.6	23.5	24.9	-1	-7.3
B. . . . .	-13.5	-4.3	-1	-3.5	14	5.6	-6	11.2	7.5	12.3	4.5	-6.9
M. . . . .	-12.9	19.3	21	-5.7	20	6.5	-14.2	4.1	20.2	24.3	1.7	-22.8
F. . . . .	-9.1	-1.4	1.9	6.8	12	12.2	0	8.5	14	9.6	6	-12.6
D. . . . .	-20	-2.4	3	6	20	26	-2	-5	38	36	-8	-20
H. . . . .	-5	9.7	22	13.2	12	10.6	-6.8	8.2	16	12.4	8	-2
Abs. shift.	90.5	66.5	109.4	96.3	162	115.2	48.3	74.6	226.2	129.3	72.2	108.9
Average . .	11.3	8.3	13.7	12.04	20.25	14.4	6.04	9.3	28.3	16.2	9.025	13.6
Relat. shift.	-46.5	50.3	107.4	77.9	162	115.2	3.9	64.6	226.2	129.3	54.2	-42.9
Average . .	-5.8	6.3	13.4	9.7	20.25	14.4	0.5	8.075	28.3	16.2	6.8	-5.4

*Schizophrenic*

The general results of this group are shown in Table VI. These are specially interesting if we consider them from the quality point of view, i.e. from the aspect and general outlook of the lines and shapes and not from the amount or direction of their shiftings. Three clear things are found in almost all the cases: (a) A tendency to lose the direction of the initial movement; (b) a tendency to reverse the movement itself; (c) clumsiness of the drawing shapes. (See Drawing No. 4.)

*Elated*

The state of elation and excitement being psychologically opposed to the state of depression and retardation, a reversion of shiftings of this group of patients might be expected if compared with those of the depressed group. This is what really happens in certain directions of the movements, as shown in Table VII. But the most impressive difference is exhibited by the results obtained with the tests of the second part of the M.P., i.e. the staircase, the chain, and the top of the castle. The drawings done by this group but not illustrated here indicate how the

ascendant and the outgoing (centrifugal or sagittal) movements are facilitated in this group of patients. The length of lines is more than twice that of the depressed group. Differences in load are also noticeable.

TABLE VI.—SCHIZOPHRENIC.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.
Males—	↙		↘		↙		↘		↙		↘	
A. . . . .	3.3	8	-13.7	3.4	-12.5	-3.4	-6.5	-6.5	5	4.5	-3.2	-3
G. . . . .	-29	-2.5	22	2.9	-0.9	-2.2	-11.8	-3.9		7	12	6
S. . . . .	-3	17.1	2	-2.8	10	0.6	25	0	-8	-2	21	0
M. . . . .	-2	13	37	18	36.2	26.4	6	15	50	3.6	26.2	33
B. . . . .	12.6	-4	-4.1	24	21.4	19.7	8.8	14	6	40	11	2.3
G. . . . .	12	4.2	8.7	7.3	4.3	6.6	18	-11	50	3.6	-1.5	27.5
F. . . . .	-4.2	1.8	1	8.1	-3.7	-3.6	13.5	7.3	0	25	16.2	-9
C. . . . .	30	4.5	6.5	-19.8	17.2	16.8	0	-15	13	5.4	1	5
R. . . . .	-18.2	-11.5	9.6	16	-3	-4.9	3	10.5	-3.2	7.3	1.8	0.6
L. . . . .		19.3		-8.6	8.2	12.4	-17	-1.5	4.5	-2.5	-9.8	4
J. F. . . .	-24	-12.6	13	-4.5	6	-6.6	-1	13.8	-9	18	-8	-21
A. F. . . .	5.2	14.6	5.8	9.6	10.6	2.4	-3.8	10.4	-4.7	-0.8	10.7	2.8
Females—												
G. . . . .	0.8	8.4	3.6	22.1	8.4	12.8	4.6	5.2	6	4	-3	-9
M. C. . . .	-23.8	0.2	-9.5	13.8	-8.9	-4.7	15.7	10	-5	6.7	6.5	-2.2
E. M. H. . .	16.3	16	1.5	4.3	11	23.3	-5.5	-4.1	2.8	5.5	6.9	-5.9
Ll. T. . . .	-14.3	-3.3	-4.3	-8.8	21.2	14	-7.8	-1.2	1.3	-4	2.8	3.6
Abs. shift.	198.7	141	142.3	176	183.5	158.4	148	129.4	168.5	143.9	141.6	134.9
Average . .	13.2	8.8	9.4	11	11.4	9.9	9.2	8	10.5	8.9	8.8	8.4
Relat. shift.	39.3	72.8	98.1	85	125.5	109.6	41.2	43	108.7	125.3	90.6	34.7
Average . .	2.6	4.5	6.5	5.3	7.8	6.8	2.5	2.6	6.7	7.8	5.6	2.1
							L.	R.				
Total average abs. shift.							10.4	9.1				
Total average relat. shift.							4.4	4.7				
C.C. = 0.47												

TABLE VII.—ELATED PATIENTS.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.
	↙	↘	↙	↘	↙	↘	↙	↘	↙	↘	↙	↘
M. A. F. . .	0	20.2	-8.4	19.6	-2.7	-5.1	12.7	21.5	-0.8	-8.6	29.1	15.8
S. . . . .	-3	17	2	-2.8	9.6	0.6	25	0	-8	-2	25	0
S. . . . .	11	-0.8	-3.6	1.6	-2	0	-0.1	8	-7	-0.5	12	14
H. . . . .	-3	-7	-11.5	-9	-4.3	1.2	18	9.4	4	-13.5	8	6.7
K. . . . .	0.3	9	1.3	6.1	-8	-2	8.6	12.6	-9	-6	29	24
H. . . . .	3.6	-4	-8.6	12.3	1.3	3.2	28	27	0	-22.5	44	17.5
M. . . . .	-13	9.5	17	-0.2	5.8	-4	4	6.8	-0.8	-2.5	20.5	8.5
Abs. shift.	33.9	67.5	52.4	51.6	33.7	16.1	96.4	85.3	29.6	55.6	167.6	86.5
Average . .	4.8	9.6	7.5	7.3	4.8	2.3	13.7	12.1	4.2	7.9	23.9	12.3
Relat. shift.	-4.1	43.9	-11.8	27.6	-0.3	-6.1	96.2	85.3	-21.6	-55.6	167.6	86.5
Average . .	-0.58	6.2	-1.6	3.9	-0.04	-0.87	13.7	12.1	-3.08	-7.9	23.9	12.3
							L.	R.				
Average length							98.85 mm.	82.15 mm.				
Total average abs. shift.							8.8	8.6				
Total average relat. shift.							7.6	7.2				
C.C. = 0.80												

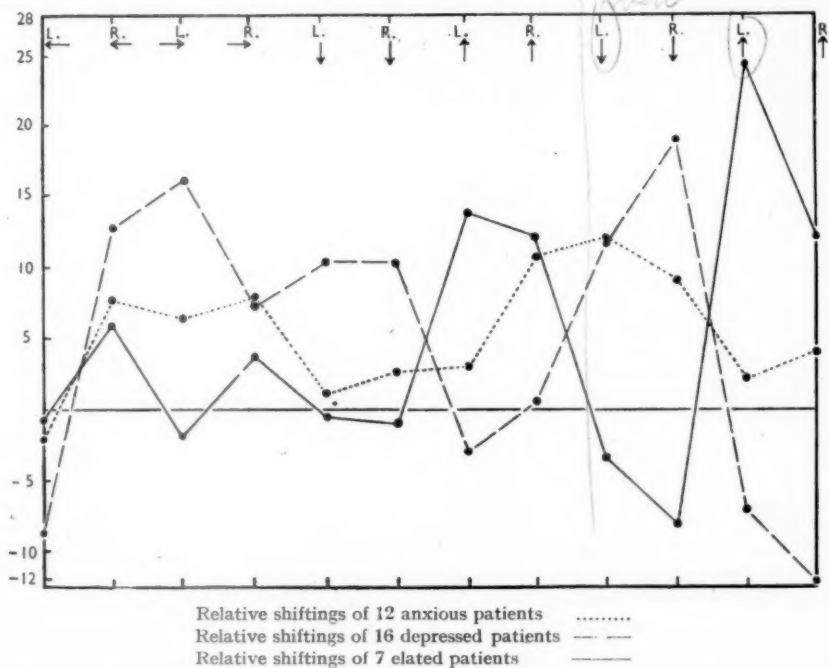
A very interesting fact is that the positive shifting in the verticals going up is much greater in the left than in the right hand, while the reverse happens with the verticals going down in the depressed group. As the left hand is subject to much less



## Discussion of the Results

In order to inspect the results of the different groups it is convenient to examine the tables where the total averages of the absolute and relative shiftings have been assembled. Looking at them and at Graphs I, II and III it appears that they cannot be either the product of chance or of purely peripheral (non-psychological) factors, such as those involved in the neuromuscular co-ordination of the implied movements.

Even if we consider these values from a purely mathematical point of view, we can see that their distribution and relative frequencies are not the result of a single general factor nor are they haphazard. In other words, the abnormally high



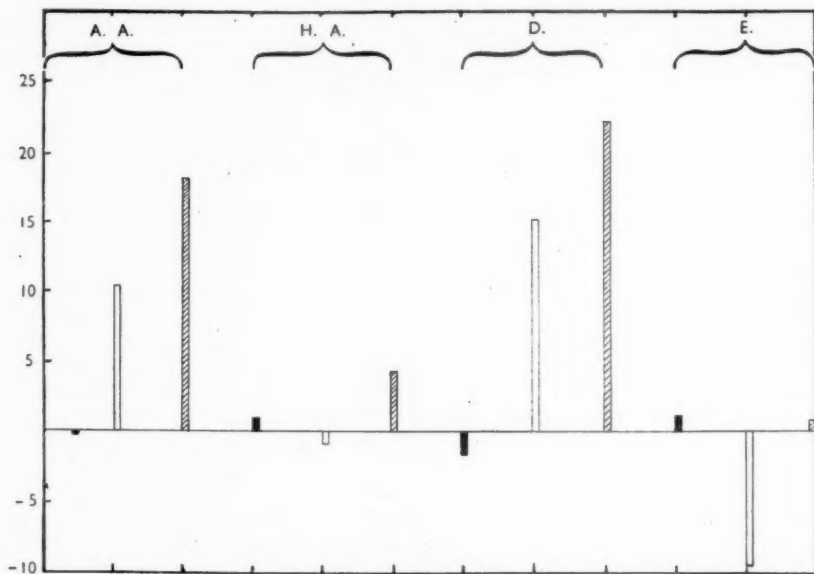
GRAPH I.—To show the "bipolar image" of the opposed groups of depressed and elated patients and the intermediate position of the anxiety group.

incidence of certain values and the unexpected absence of some others in each consistent group—as compared with the rest—point out that there must be *special* reasons which explain the particular distribution of the shiftings among each group.

If we now consider which are the attitudes of reaction that we can expect to find among the subjects of these groups, according to their peculiar state of mind, we will see that whereas a given set of conative trends can be asserted from the clinical picture of a set of subjects we find a corresponding set of prominent shiftings in its output in the test.

Graph III shows that the extreme values of relative shiftings are successively obtained by different groups of subjects and, precisely, by those which according to

the peculiarities of their clinical picture ought to possess the attitudes of reaction to which these shiftings seem to correspond. As a matter of fact we know that in states of depression and retardation a general fall in the psychobiological energy gives rise to a diminution of the strength of conation and to a consequent difficulty of those movements which need more psychomotor energy for their performance, i.e. the movements which are directed against the action of gravity. Then we may expect that in depressed and retarded patients we must get a noticeable increase in the shiftings in the vertical plane, this increase being characterized by the ease of the descendant movements and the difficulty of the ascendant ones; we must find



GRAPH II.—Relative amount of auto-aggression (A.A.), hetero-aggression (H.A.), depression (D.), and elation (E.), as shown by the relative shiftings of:—

35 normal subjects    16 depressed patients    8 suicidal patients

very high positive shiftings in the lineal drawings and very high negative shiftings in the opposed drawings, as compared with those of the group of normal subjects and especially if compared with those of a group of elated patients (Graph III).

When we compare these results with those of the group of suicidal patients we are able to detect a difference in the results of the left and the right hand; in fact the right hand of the average depressed patient is unable to shift up in the ascendant vertical movements but the left hand sometimes does and, what is more interesting, the shifting down shown by the left hand of the suicidals is clearly greater than the corresponding movement of the left hand of depressed non-suicidals. If we look at the clinical records of these patients we shall be surprised to find that those left hands that are able to shift up and down in the vertical plane belong to patients whose previous personality is considered as having been cheerful, jolly, active, and rather elated. If we put this fact in relation with what has been said about the unconscious meaning

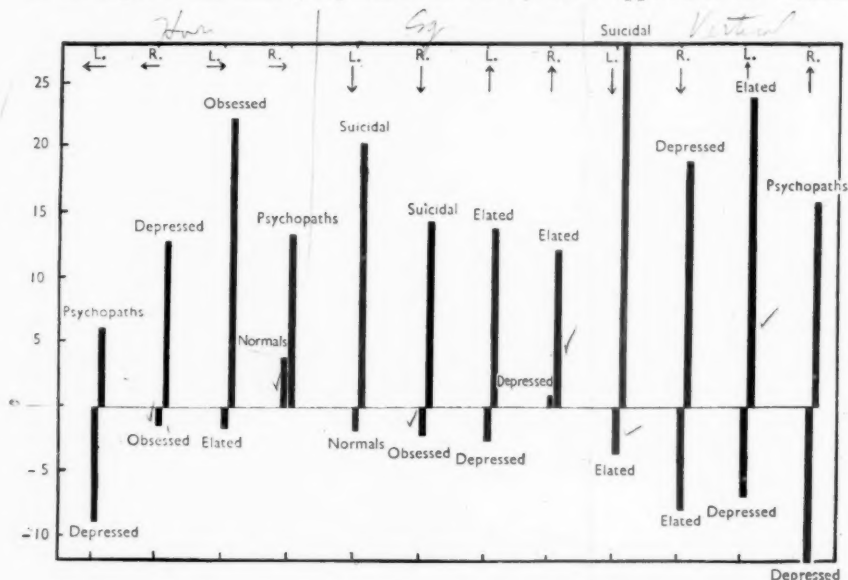
of the expressive movements in the left side of the face and the body (Werner Wolff) we can venture the hypothesis that the behaviour of the left hand is more related to the constitutional trends of the individual, the right hand expressing rather what is actually present in his mind or, in other words, the former being related to genotypical and the latter to phenotypical conative trends. Once we have thought about this, many facts confirm it; surgeons have been impressed by the facility with which untrained assistants can still handle the instruments with their left hand in the operating theatre when they fail miserably to use the right hand, owing to their emotional state. And I have recorded some cases in my experience in which the individual was unable to start the test with the right hand and did it successfully with the left. Another fact which may prove the same assumption from a different angle: if we compare the coefficients of correlation of the shiftings of the left and the right hand in the same experimental set with those of the measures of the same hand in two different sets of trials (reliability) we shall be surprised to see that there is on the average more correlation in the results of a given hand in two different sets than in the results of both hands on a given set (0.78 is the average value of the first correlation in 35 cases and 0.52 is the average value of the second correlation). It is striking to compare the enormous variability of the first coefficient which ranges easily between 0.94 and -0.45, with the second which oscillates merely between 0.62 and 0.95, thus seeming to prove, too, that each hand expresses predominantly a different set of conative trends. In order to save space we quote below only half the number of coefficients compared:—

Patient	Correlation between the left and the right hand in the first set of experiments	Correlation between the twelve relative shiftings in the first and in the second set of experiments (intervals ranging from 5 up to 21 days)
K.	0.94	0.89
G.	0.92	0.90
R.	0.84	0.86
F.	0.82	0.87
F.	0.72	0.83
J.	0.63	0.80
S.	0.55	0.89
H. B.	0.49	0.84
P. G.	0.49	0.78
T.	0.48	0.80
C.	0.48	0.81
J. A.	0.41	0.76
M. H.	0.39	0.88
M. C.	0.31	0.74
E. D.	0.01	0.82
E. L.	0.02	0.72
L. T.	0.25	0.90

The results of the second part of the M.P. confirms the two fundamental assumptions we have advanced: (a) Patients with depression, i.e. with a loss of psychomotor energy, have a tendency to go down in all their combined movements in the vertical plane; (b) this tendency is exhibited more in the left hand in the case of people who can be considered as constitutionally depressed, and more in the right hand in those whose previous personality was rather full of psychomotor energy. But as there are mixed states of depression and elation, so we find in them the coexistence of contradictory shiftings, i.e. of positive shiftings in the ascendant and in the descendant direction. These mixtures are found to be more marked in one hand or in another according to the permanent or purely actual character of the conflicting trends of personality.

Another step in the interpretation of the results comes from the analysis of the possible meaning of the shiftings in the sagittal plane. If we look at Tables IX and X where the general results of the main groups have been concentrated, we can see that the maximum of shifting in the inwards (egocipetal) direction is to be observed

in the group of suicidal patients and the maximum in the outwards (egocifugal) direction appears in the group of the elated subjects, whereas the corresponding minimum values of these shiftings (taking the averages of both hands) corresponds to the groups of elated and depressed, respectively. Thus we find again opposed in the sagittals the same group which we already found opposed in the verticals.



GRAPH III.—Extreme values of the relative shiftings in the twelve fundamental measures.

TABLE IX.—AVERAGE OF ABSOLUTE SHIFTINGS OF THE DIFFERENT GROUPS.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	Total av. abs. shift.
✓ Normals (35) ..	8.6	7.45	5	7.5	5.8	4.45	6.2	5.45	4.5	5.45	6	5.75	72.15
✓ Epileptics (32) ..	9.16	8.8	11.1	10	7.18	7	8.8	9.4	9.8	8.75	10.7	9.1	109.79
✓ Psychopaths (7) ..	9.8	15	16	17	9.9	5.1	9.9	9.35	7.66	10.6	14.1	15.7	140.11
✓ Depressed (16) ..	12.5	13.2	18.9	11.7	11.9	11.4	6.2	9.8	14.1	18.9	8.4	13.3	150.3
✓ Anxious (12) ..	8.1	10.7	12.7	10.35	9.7	8.6	10.5	11.7	14.3	12.4	9.4	8.8	125.25
✓ Schizophrenic (16) ..	13.2	8.8	9.4	11	11.4	9.9	9.2	8	10.5	8.7	8.8	8.4	117.3
✓ Suicidal (8) ..	11.3	8.3	13.7	12.04	20.25	14.4	6.04	9.3	28.3	16.2	9.02	13.6	162.45
✓ Obsessed (6) ..	5.3	9.7	22.2	13.2	11.8	10.3	8.3	11.3	8.4	7.7	6.66	8	122.86
✓ Elated (7) ..	4.8	9.6	7.5	7.3	4.8	2.3	13.7	12.1	4.2	7.9	23.9	12.3	110.4

TABLE X.—AVERAGES OF RELATIVE SHIFTINGS OF THE DIFFERENT GROUPS.

	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.
Normals ..	-3.94	4.25	1.35	3.8	-1.7	1.2	0.39	1.65	-1.27	-1.94	3.05	-0.9
Epileptics ..	-4.5	5.42	8.62	5.5	2.74	2.75	0.8	4.2	5.3	4.75	6.6	2.4
Obsessed ..	-6	-1.4	22.2	13.2	11.8	-2.1	4.9	8.3	0.8	2.4	3.7	-2.7
Anxious ..	-1.85	7.9	6.5	8	1.1	2.9	3.1	10.8	11.7	9.2	2.3	4.4
Depressed ..	-8.7	12.8	16.1	7.5	10.4	10.4	-2.6	0.75	11.3	18.9	-7	-12
Schizophrenic ..	-2.6	4.5	6.5	5.3	7.8	6.8	2.5	2.6	6.7	7.2	5.6	2.1
Suicidal ..	-5.8	6.3	13.4	9.7	20.2	14.4	0.5	8.07	28.3	16.2	6.8	-5.4
Elated ..	-0.58	6.2	-1.6	3.9	-0.04	-0.87	13.7	12.1	-3.08	-7.9	23.9	12.3
Psychopaths ..	6.1	10.5	9.9	13.5	4.8	0.55	7.5	9.2	7	6.7	12	15.7

How to elucidate the real meaning of these shiftings? From a purely speculative point of view the positive shiftings outwards could be interpreted as a sign of (a) Extraversion, (b) hetero-aggression, or (c) liberality; those in the inwards direction being then considered as a sign of: (a) introversion, (b) auto-aggression, or (c) selfishness. In any case, this direction, i.e. inward and outward in the sagittal plane, seems to be the most fundamental for expressing the relation between the subject and the external world, especially the social world (since our movements in that direction are mainly concerned with reactions towards human beings). I cannot reproduce here how I have to come to the conclusion that from these three possibilities the second, i.e. auto-aggression, seems to be the more correct. Nevertheless, some of the reasons I had for assuming this must be quoted: (i) There is no doubt that the auto-aggressive tendency must be the one more prominent among suicidal, but there is no reason to believe that it must be so prominent among schizophrenic patients. Then the big shifting in the inward movements of the first and the slightness of it in the second can be explained. Were we to admit, however, that these shiftings are a measure of introversion we should come to the conclusion that there is more introversion in the suicidal and depressed patients than in the schizophrenics, which is not true in practice. (ii) We know how much the average obsessed is afraid of anything that may hurt his personality, thus showing, so to speak, a conscious reluctance to auto-aggression, whereas we find an increase of hetero-aggressive trends among the groups of psychopathic and anxious personalities; these facts are confirmed by our findings, if our assumption is proved to be justified. (iii) Considering the personal trends of character of the group of normals, whose different individuals are well known, and ranking them from the three points of view under discussion, we easily found that the amount and direction of their shiftings closely correspond to the amount of their auto- or hetero-aggressiveness, whereas there is no definite relation with the rest of the supposed trends. Then the coincidence of a maximum of shifting of the groups of suicidal and depressed patients in the lines going down and inwards and a maximum of shifting of the elated in the lines going up and outwards merely means that in the average of depressed and suicidal there is a coincidence, too, of depressive and auto-aggressive trends, as there is a coincidence of elative and hetero-aggressive trends in the elated. But these coincidences are not absolute; we can easily imagine a depressed patient with hetero-aggressive trends or an elated patient with auto-aggressive trends. In fact, the correlation (obtained by Miss Wood) among both kinds of shiftings in the group of 16 depressed was merely 0.28 with the right hand and 0.60 with the left hand. In a case where the depression was apparently produced by misbehaviour of the patient's wife we found a very high positive shifting of the outward movements of the right hand, the patient admitting later that he had the intention of killing the lover; so many other particular cases could be mentioned in which all possible combinations between the sagittal shiftings are to be observed, always in accordance with their individual peculiarities of mental constitution and situation. In a given individual the oscillation of his shiftings in the sagittal direction can thus give account of the variations of what we could call his "vital space of direct action", i.e. the expansion or retraction of the zone in which he is trying to act (the term "aggression" is used in its psychological sense).

The interpretation of the horizontal shiftings is far more complicated, and I confess that I am so far unable to advance any suitable explanation of them. It is really amazing how in spite of these directions of movements being more easily controlled by the individual's will and experience, the normal group exhibit in them an average of total shifting which is superior to those exhibited in the sagittal and the vertical planes. Some cues could be obtained, for advancing a tentative hypothesis, from the most significant shiftings in this plane, such as, for instance, the one of the left hand of the obsessed (when moving towards the right side), but I shall refrain from any premature attempt of this kind.

The main objections that can be raised against our interpretation of the results may be: (a) The relatively small number of cases on which it is based; (b) the insufficient statistical control of the fundamental measures; (c) the lack of control experiments with other alternative techniques. Therefore my results are preliminary, but, those already commented upon are promising enough to stimulate further investigations upon this line. I have started some new experimental research in this field, asking the subjects to accomplish the movements not only without visual control but under the influence of distraction; I have devised, too, a new apparatus (which I call the "axistereometer") enabling the exploration of conative trends in function of the changes in the measure of a given distance in all possible directions of the space; I have even started to investigate how the appreciation of the lineal movements in the skin (by "local signs") is influenced by the attitude of reaction of the patient. All this means, of course, that we are still at the entrance of the new field and cannot forecast its ultimate boundaries.

#### CONCLUSIONS

In spite of the limitations of our work, we feel that the following conclusions may be drawn:—

(a) When a subject is asked to accomplish a set of lineal movements in the three fundamental directions of space, according to the technique explained in this paper and described as "myokinetic psychodiagnosis", a collection of data may be recorded that provides interesting cues for detecting the conative trends of his personality.

(b) The data recorded are quantitative and qualitative, and according to our experience can be considered as reliable and reciprocally complementary. Among them, the differences between the corresponding measures in both hands are relevant to indicate the degree of cohesion of the personality or, in other words, the amount of coincidence in its constitutional (permanent) and its actual (transitory) attitudes of reaction, the firsts being more connected with the results of the left hand (except in left-handed individuals) and the seconds with those of the right hand.

(c) The results obtained in different groups of normal and abnormal subjects point out that the average of absolute shiftings is a good rough measure of what is called "nervous equilibrium", but that the averages of relative shiftings, especially in the sagittal and the vertical planes, are far more psychologically significant.

(d) There are reasons to believe that an objective measure of the amount of depression or elation of a given individual can be expressed in function of the sign and value of his relative shiftings in the vertical plane, and that the amount and direction of its aggressiveness can be expressed and evaluated in function of the sign and amount of his shiftings in the sagittal plane, thus providing a good index for detecting the danger of suicide or hetero-aggression.

(e) If more systematic research is carried on in this field and a complete statistical analysis made of the results, thus providing the opportunity for its standardization, this technique may afford unexpected and promising results not merely for the psychiatric diagnosis but for the prognosis as well, thus supplying at the same time an objective measure of some of the most important of its elements.

Owing to the kindness of the Society for the Protection of Science and Learning, which allowed me a grant for research, and of Prof. Mapother who was willing to let me handle the clinical material of the Maudsley Hospital during the past six months, I am able to present some preliminary results of this technique which are rather encouraging. I must express my thanks to all the medical staff of that Hospital and especially to Dr. Aubrey Lewis for his kindness in revising this paper.

[Note: The accuracy of the figures in the tables cannot be vouched for. Though the evaluation of the data is clear, the author's typing errors make the arithmetic difficult to correct, as his original figures are not available.—ED.]

## Section of Otology

President—T. RITCHIE RODGER, M.D.

[December 1, 1939]

### DISCUSSION ON THE TRANSMEATAL APPROACH TO THE MASTOID

**Dr. G. B. Brand:** Bruno Kecht, in a paper published in 1936, gives a detailed account of the history of the operation since Thiess Senr. first practised the transmeatal approach thirty years ago, and also describes the results of the operation which he personally observed in 49 cases.

The transmeatal method has found little favour in this country, but since Lempert<sup>1</sup>, in May 1938, published a paper on the "Endaural, antauricular surgical approach to the temporal bone, with a summary report of 1,780 cases", interest has been aroused and many otologists have been practising his method.

Lempert claims that his approach is suitable for any operation on the temporal bone, including all forms of complication. Of his 1,780 operations, 1,595 were done for acute mastoiditis. The technique is fully described in his article, but for those who may be unacquainted with the procedure, the purpose of the incision is to make a large membranous window without interfering with the cartilage of the auricle. Lempert describes a little triangle situated between the helix and the tragus which he calls, "the antauricular suprameatal membranous triangle", and claims that the surgical use of this space makes possible the endaural approach in all attacks on the temporal bone. He considers that the advantages gained more than justify his abandonment of the postauricular approach.

Preliminary infiltration of the incision areas with 1:10,000 adrenalin solution, usually ensures a fairly bloodless field; the membranous window is formed by making three incisions down to the bone, and removing and discarding the resulting flap of soft tissue, including the periosteum.

For the *radical operation*, the first incision begins on the postero-superior wall of the meatus immediately outside the postero-superior attachment of the drumhead to the tympanic ring, and is carried downwards and outwards to the lower anterior border of the concha. The second incision starts at the same point as the first one, and is carried outwards along the postero-superior wall till it reaches to the base of the antauricular suprameatal triangle into which it passes just above the cartilage of the tragus. The third incision joins the outer ends of the first two, starting at the first and passing into the suprameatal triangle, just inferior to the helix to join the second incision at the apex of the triangle. After the flap has been removed and discarded, it is very easy to elevate the attachments of the auricle to the temporal

<sup>1</sup> *Archives of Otolaryngology*, 1938, 27, 555.

bone, until the auricle is freely mobile. The insertion of a mastoid retractor then gives an adequate exposure of all the landmarks.

If a simple cortical attack is required, the first two incisions begin at the junction of the bony and membranous parts of the postero-superior wall, and if one wishes to explore the petrous part, a fourth incision is made after the radical ones, and a flap removed from the bony part of the anterior wall. Should it be necessary to attack the squama, the junction of the incisions at the apex of the antauricular triangle can be prolonged upwards in front of the auricle.

#### *Advantages Claimed for this Method of Approach*

Exposure is obtained with much less sacrifice of tissue. No muscle tissue or cartilage is injured. Artery clamps are not required. The endaural window gives a fuller and more direct view of the surgical field, and more ready accessibility, especially in the case of the radical operation, and the petrositis operation. It also reduces by about 1 in. the depth of the surgical manipulations on the bone.

After the bone operation is concluded, no stitching or packing of the wound is necessary. A superficial dressing is worn for four days only. There is a constant unimpeded drainage of the osseous wound throughout the period of healing, and constant visibility of the osseous wound. The period of healing and convalescence results in the least possible social and economic inconvenience to the patient.

The endaural wound cannot be closed and will not close of itself until the mastoid wound is fully granulated. When the wound is healed the meatus in appearance very closely resembles its condition before operation. The scarring is stronger and firmer and there is less tendency to reinfection.

It is also claimed that hearing is better.

#### *Cases Operated on by the Lempert Method*

Since October 1938 in the Ear Department of the Victoria Infirmary of Glasgow, we have operated on two acute cases (simple cortical type) and 23 radical cases, by the Lempert method of approach. All the cases were straightforward and uncomplicated.

*Acute cases.*—Of the acute cases, the first was a young woman of 31 years, and the other a boy of 6 years.

With regard to the acute cases, we have no great urge to alter our method as the ordinary Schwartze, with primary closure of the wound, except for a thin gauze drain from the antrum to the lower part of the postauricular incision, has proved so uniformly successful that the average case usually leaves hospital in just over two weeks from the time of operation.

The two cases by the transmeatal approach were not so satisfactory. Both were done towards the end of the series. In the case of the young woman, the approach was satisfactory and the bone operation was completed without difficulty. Almost immediately afterwards there was a complaint of pain in the mastoid region without rise of temperature. This became increasingly worse in spite of a free serous discharge from the meatus, and was only relieved after about ten days by making an incision over the mastoid tip region, which released a large quantity of seropurulent discharge. Patient is still in hospital (five weeks) but is now healing well.

In the case of the child aged 6, approach and bone operation were also satisfactory. The convalescence was free from pain or discomfort, but the discharge persisted for four weeks. In four and a half weeks there was perfect hearing, and a more or less normal meatus, with a small scar in the antauricular triangle as the only obvious sign of surgical interference.

*Radical cases.*—Coming now to the radical cases, there has been no difficulty in making the approach, and the radical operation has been simplified. When the bone operation is completed, there is no stitching or packing of the meatus or wound, and a simple superficial dressing is applied. This is changed daily for four days, and the bandage can then be discarded. Healing goes on steadily, and the granulations can be watched and kept under control with silver nitrate applications where and when necessary. In many of the cases there has been perfect epithelialization in five and six weeks, and practically no discomfort in convalescence.

There is much less shock following on the operation. Patients have no complaint of pain or stiffness of the neck, and are generally up and about on the fourth or fifth day. When healing is completed, the meatus is usually of good calibre, though several cases show narrowing, and our impression is that the hearing results are better than they would have been after a postauricular incision.

One of the cases had a double radical, with a postauricular incision on one side, and an endaural on the other. The hearing defect in both ears was as near as possible equal before operation, but, after healing, there was an undoubted improvement on the endaural side. This may not mean anything, but it helped to confirm the impression that hearing is better after an endaural incision.

In our early cases we followed Lempert's technique meticulously. We did not find the burr entering the antrum without a good deal of burring, and as a result, in several cases we had transient facial paralysis, and somewhat alarming labyrinthine irritation, the result of overheating of the bone. If the burr is used, care must be taken to avoid overheating, by making its action very intermittent.

One must also bear in mind the new orientation of the approach, when one has been long accustomed to operate on the tympanic region from behind forwards. The approach is a good one, and gives an adequate exposure. It seems to be a great advantage in cases of radical mastoid operation, especially in the sclerotic types. It is the ideal method of approach for an attico-antrotomy. It is certainly well worth trying, and when an increasing number of cases is done, we may be able to endorse all that Lempert claims for it.

**Mr. Walter Howarth** said that this transmeatal approach was not by any means new. Indeed, such an approach to the mastoid was by a long way the oldest method of approach, and Kessell and his pupil, Hoffman, employed it very largely. It was a recognized method of opening the mastoid. The incisions made in the posterior meatal wall were very much like those of the ordinary Körner flap, the mastoid was exposed in that way by these earlier workers, and it was really only when Zaufal and Stacke described two rather separate methods of opening the mastoid from behind that this transmeatal approach fell into abeyance. It was revived again comparatively recently when local anaesthesia came into use in Germany, and von Eicken and Gompertz and others employed it very largely. When local anaesthesia was found effective for the post-auricular route the approach was given up again and the mastoid opened from behind under local anaesthetic.

One of the difficulties of the transmeatal approach until recently was the width of access to the various portions of the temporal bone and it was perhaps because of this that it was used only in a limited type of case. However, the technique that has been perfected by Mr. Lempert in New York did away with a good deal of that difficulty by providing a much freer access to the mastoid.

As to the method of opening the mastoid a sharp dental drill could be used, and if it was directed inwards and slightly backwards, the antrum lying only about 1 to 2 mm. below the surface was easily entered. Then the opening backwards could be enlarged with burrs and curettes towards the spine of Henle and

downwards, and so extend to the whole area. Of course the great thing was the mobility of the auricle that this method afforded. One could move the whole auricle away from the mastoid and expose not only the zygoma, but almost the tip and pull it back. It was quite extraordinary what traction would do.

His own view was that no set operation ought to be used of necessity in every case, and in point of fact this transmeatal approach was not altogether suitable—certainly not in his own hands—for extensive infection in an acute mastoiditis. He personally set great store by the retrofacial group of cells leading down to the large cell at the tip of the mastoid process, and he was accustomed to take away the tip in the majority of cases until the digastric nerve was exposed. It was sometimes very difficult indeed by this method to get to the tip at all. There was also a group of cells behind the sinus which might extend a considerable distance, and it was not always possible to be certain of an adequate exposure in that area of the temporal bone. In some cases he had not been satisfied with this approach from the operative point of view, and when it seemed desirable he had made an approach by a postauricular incision as well. This did not vitiate the principle of the after-treatment of the case, except that, of course, one did have this auricular incision which was immediately sutured. The great advantage, it seemed to him, in acute cases, particularly in children, was that one had, of course, a very free drainage through this triangular gap in the posterior nasal wall and one had no packing with its consequent distress for the patient. There was continuous draining, the granulations filled up the cavity and the posterior meatal wall epithelialized over, and the contour was much the same as before the operation. That was the great advantage in children because they were rather terrified of the mastoid dressing, particularly if the postauricular wound was left open.

In the modified radical operation this method was particularly useful, as one was much nearer one's objective with this method, and could open the tympanum and see round the attachment of the drum right into the aspect of the tympanic cavity. One could also if necessary remove the head of the malleus to facilitate drainage from the anterior cleft.

**Squadron-Leader G. H. Bateman :** In May 1938 I saw Dr. J. Lempert of New York operating on the mastoid by his endaural approach and on my return to England I practised the new technique of bone surgery on some temporal bones. In July 1938 I began to use this approach for both simple and radical mastoid operations. Since then I have done 32 simple mastoid and 21 radical or modified radical operations using the endaural approach. This series has convinced me that the end-results of this operation are at least as good as the results I have obtained with the postauricular incision and that therefore factors other than results may be allowed to influence one's choice of operations.

In the simple mastoidectomy the endaural approach makes the operation technically more difficult, but, with patience, the operation can be done completely in every case so far encountered and the resulting increase in comfort for the patient makes the extra trouble worth while. The after-treatment is less tedious because packing the cavity is done away with, whilst retaining the advantage of preserving direct inspection of the cavity.

In the radical operation the incision gives a better exposure of the middle ear than the postauricular incision; though one must adopt a different approach to the antrum, as it is impossible, with the limited exposure, to bevel the edges of the bone excision as is done in the postauricular operation. Thus one opens the antrum through its anterior wall, i.e. the postero-superior meatal wall, as is explained in Lempert's paper (*Arch. Otolaryng.*, 1938, 27, 555).

I did at first use Lempert's method of after-treatment of the radical mastoid cavities without any packing but I found it impossible to keep a sufficiently wide external auditory meatus by his methods and have reverted to packing the mastoids. Therefore the reasons for using this approach are different in the two types of mastoid operations. In the simple mastoidectomy I use it because the after-treatment is less painful and the cosmetic result is excellent. In the radical I use it because the exposure of the middle ear is better and the bleeding is considerably lessened and the cosmetic result is good.

The results of operations :—

	Operations	Unsatisfactory and Moist
Simple mastoidectomy . . .	30	4
Radical or modified radical mastoids	21	— 5

So far in no case has an unsatisfactory result been obtained "because I have failed to open up the mastoid air cells completely. If a patient is anxious to get back to work soon after the operation he is able to do so much sooner with the endaural incision because he can dispense with the bandage after the first ten days.

I had more difficulty in settling down to a routine for after-treatment of the radical mastoids and modified radical mastoids because I started by following Dr. Lempert's technique and was considerably bothered by narrowing of the meatus in the first few cases. However, by packing the cavity for the first fortnight, this difficulty has been overcome and the results have been very satisfactory since then. I obtain a much better view of the attic of the middle ear with this approach and stimulated by Lempert's excision of the head of the malleus in his operation for otosclerosis (*Arch. Otolaryng.*, 1938, 28, 59) I have in several cases of attic suppuration excised the head of the malleus with very gratifying results.

As a result of my experience with these 53 operations I am satisfied that the endaural incision for mastoid operations, as described by Dr. Lempert, gives a perfectly adequate exposure and is the incision of choice for mastoid operations. But it does require a different technique of bone surgery and the application of the ordinary technique to this incision will lead to disappointment with the results, because the air cells have not been completely removed and this approach will therefore be abandoned. The operation must be made relatively bloodless or the difficulties are greatly increased. This can be done by the injection of novocain and adrenaline ten minutes before operation and by the use of adrenaline or bone wax on bleeding points in the bone.

**Mr. John Gerrie :** The transmeatal approach to the mastoid has been my routine procedure for the past six months. I do not open the mastoid antrum directly, as advised by Lempert, but as if one was approaching it from the post-aural route. I have never had difficulty in removing cells, and I have exposed dura of the middle fossa and the lateral sinus in acute cases as a routine.

In complications, there seems to be no difficulty in obtaining access to the middle or posterior fossa or labyrinth. I have had four cases of perisinus abscess, but no lateral sinus thrombosis. A case of circumscribed labyrinthitis and facial paralysis showed the rosette of granulations on the horizontal canal and of the fistula into the facial canal. In a case of diffuse purulent labyrinthitis, the labyrinth was opened with ease.

*After-treatment.*—At the end of the operation I introduce some streptocide cream into the mastoid cavity from a soft gelatin capsule. This accelerates healing of the intrameatal wound and when the cotton-wool pledget is removed from the ear next day, the patient experiences no pain.

Some cases, with profuse discharge, require mopping out every four hours for the first day or two. If the ear is not drying, syringing with sodium bicarbonate solution is employed. Spirit drops are too painful for the first day or two. After three or four days all dressings, save a piece of cotton-wool, can be dispensed with, and the patient allowed up. In the case of acute mastoids no packing should be used, but in the case of modified radicals or radicals, it is advisable to put a small B.I.P.P. pack into the region of the aditus, otherwise stenosis may occur.

**Results.**—When a cortical operation was performed, the average healing time for the intrameatal wound was thirteen days, and the average length of stay in hospital was fourteen days. This compares very favourably with the results of the post-aural operation. At the time of discharge all the middle ears were dry, but two cases came back with moist middle ears due to nasopharyngeal sepsis.

**Advantages of this method.**—(1) It is the method *par excellence* in children, as the dressings are fewer and easier and there is no pain.

(2) The period of stay in hospital is much reduced.

(3) The method allows of easier access to the middle ear and attic.

(4) The cosmetic result is perfect.

(5) There are no complications connected with a post-aural wound, e.g. breaking down of wound or the formation of a post-aural fistula.

**The President** said he thought that the first question would be the facility of approach as compared with the usual methods and, secondly, the complications which were present in so many cases. The late J. S. Fraser and he found that they met almost identically the same proportion of intracranial complications in their mastoid operations. Mr. Fraser's figure was 10.5% and his own 10.6%. If there were 10% of intracranial complications in routine mastoid work then obviously it was of importance that this new approach gave every facility for dealing with the complication. It had always been laid down that the proper procedure, if part of the dura was exposed by infection, was to remove the sinus plate or the dural plate until the area of pachymeningitis was surrounded by healthy dura so that there could be no collection of pus between the dura and the bone. One would like to be satisfied that the method described offered adequate opportunity for dealing with such conditions.

**Mr. J. F. Simpson** said Mr. Cecil Graham at St. Mary's Hospital had used this method of approach, though with different minor details, as a routine some ten years ago. His own impression was that if applied as a routine it was not a satisfactory procedure. Mr. Simpson did not like the vertical strokes of the mallet and gouge thus necessitated, though the use of hand gouges and mechanical burrs might overcome this. The extreme traction sometimes applied to the auricle to improve the view seemed unsurgical when a post-aural incision would have provided adequate exposure.

Generally, healing time did not appear to be shortened nor did dressings seem to be less uncomfortable. Lateral sinus thrombosis had been successfully dealt with by Mr. Graham using this approach. Mr. Simpson, however, had given up the method for the last six years.

**Mr. W. I. Daggett** said that in the first two cases in which he carried out Lempert's method he left no internal dressing, and in both of them the cavity filled up with blood-clot. He tried to remove that blood-clot, but found it extremely difficult. Finally, he syringed it out with a catheter. He had never used a burr, but he had found no difficulty in obtaining the approach he wanted. Two of his cases showed a rather narrow meatus afterwards, but the case-notes showed that in both the meatus

was narrow before treatment started. The beginner, fearing a cramped field, was apt to cut too near and encroach upon the conchal cartilage.

**Mr. Watkyn-Thomas** said that the cells most easy to miss were those in the zygomatic root, in the apex, where the tip cell was often double, and in the region of the petrous angle where the lateral sinus turned back. He noticed in the model which had been handed round there was a group of cells unopened at the apex.

Painful dressings could be avoided by free use of sterilized vaseline or by rubber dam packing. He found little pain in after-dressing. The greatest difficulty in the radical operation was the contraction of the cavity, and this new method did not safeguard against such contraction.

**Mr. C. A. S. Ridout** felt that if this method were adopted as a routine in the treatment of acute mastoids, not only would the very extensive ones not be done satisfactorily, but a great deal of time would have to be added to the operation. He believed that it would make each operation a quarter of an hour longer or more on the average, and this was a serious consideration when hundreds of cases came forward in the course of the year. It was a disadvantage against which there would be no corresponding value. The cells over the lateral sinus and the petrous angle were very difficult to deal with. He could not see that this method offered any advantage over the existing technique for acute mastoiditis, but for the modified radical operation there might be some advantage.

**Mr. Scott Stevenson** said that the object of a mastoid operation was to open up all the cells in the mastoid area. In spite of the experience of those who had carried out this operation, he could not believe that this route gave access to all the different mastoid cells, though he believed there was a place for it in the modified radical operation. All of them disliked the classical radical operation and had tried out other methods. There was probably a place for the Lempert technique here.

**Mr. Terence Cawthorne** said that when he read Dr. Lempert's paper he had not considered trying what seemed to be a difficult approach. Later he resolved to give it a trial both for the simple and the radical operation. In two out of the three acute cases the after-treatment seemed to be more painful than when the post-auricular approach was used. This may have been due to a technical fault, whereby he had encroached upon the cartilage. With regard to the radical operation, he had soon found that the anterior approach was easier and quicker than the post-auricular one. He had not found any difficulty in using the ordinary gouge, although of course if was necessary to become accustomed to an altered cutting angle.

The after-treatment had called for more attention and care than after the ordinary radical mastoid. By packing the meatus and cavity with some iodoform gauze in an oiled-silk bag the dressings were painless.

He asked Squadron-Leader Bateman if he would use the method in such complications as a brain abscess? A further question he desired to ask was how much cortex was removed in the operation?

**Squadron-Leader Bateman**, in reply to Mr. Cawthorne's question, said that all the cortex was removed; there was no overhanging bone. All diseased bone was taken away—that, indeed, was essential. The zygomatic group of air cells was more easily approached by this incision than any other group; the tip cells were not so easy, and the marginal cells behind the lateral sinus did present more difficulty, but this could be done. It took more time with this incision in the acute mastoid,

but unless all the diseased bone and all the overhanging edges were removed there was likely to be trouble afterwards.

In the course of an operation he had exposed all the semicircular canals, and had reached the facial nerve. He had decompressed the facial nerve without any more difficulty than he would have had with a post-auricular incision, and he had opened the superior semicircular canal with this incision. He agreed with other speakers that the radical mastoid did not take more time, but the cortical mastoid certainly did.

**Dr. G. B. Brand** (in reply) said that he had not yet felt bold enough to attack an acute mastoid with complications by this route. However, he did not agree with speakers who had said that the approach could not be adequate. In his view the approach was adequate enough but he did not feel that the drainage for the first four or five days was as good in the simple mastoid operation as it was with the post-auricular wound. It might be that time would alter his opinion.

## Section of Neurology

President—GEOFFREY JEFFERSON, M.S.

[December 7, 1939]

### The Radiological Diagnosis of Chronic Subdural Hæmatoma

By J. W. D. BULL, M.B., M.R.C.P.

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THE primary object of this communication is to attempt to demonstrate the value of neuroradiology in the elucidation of the diagnosis of subdural hæmatoma. The condition has been recognized for many years, and is now becoming more frequently diagnosed.

*History.*—The earliest case I have been able to trace was described by Burrows in the Croonian Lectures for 1835. A man, aged 53, was admitted to St. Bartholomew's Hospital in October 1834, under Dr. Latham.

"The man stated that his mind had been a good deal oppressed for a twelvemonth, in consequence of adversity, and that six months prior to his admission he had been attacked with headaches which were relieved by blisters. About a month before admission, headache and giddiness returned which prevented him from following his occupation. Upon his admission he complained of giddiness with inability to retain his urine or fæces. He was cupped from the temples and blistered; but his mental faculties gradually declined and he remained in a state of insensibility approaching to coma; without any material change he lingered a few days and died."

The post-mortem examination is carefully described and the lesion was clearly a chronic subdural hæmatoma. Burrows said: "The left hemisphere of the brain was much compressed by the distended sac of blood and coagula; the left ventricle was small from pressure; there was nothing further remarkable in the substance of the brain." The specimen was placed in the St. Bartholomew's Hospital Museum.

In 1845 at this Society Sir Prescott Hewitt, the Curator at St. George's Hospital, delivered a masterly account of the lesion. He described what is probably the first published bilateral case. It was in a man aged 51, with a history similar to that of Burrows' case. In neither author's material was trauma mentioned in the history and no fracture of the skull was found at autopsy.

It would not perhaps be an altogether idle speculation to suggest that had not Sir Benjamin Brodie shown so much reluctance about opening the dura, for he found that all such cases went septic afterwards, he might well have been the first to cure cases of subdural hæmatoma. He was after all a colleague of Sir Prescott Hewitt's, and was well versed in the science of the cranial surgery of the time.

In 1905 Bowen made an important contribution, collecting 72 cases from the literature including one of his own.

In 1914 Trotter described four cases upon which he had operated. He was perhaps the first of the more modern authors to stress the importance of the disease. He stated that the lesion probably frequently escaped clinical recognition, and he made a plea for establishing the diagnosis in the early stages of the disease, for he said cure would then result in every case.

\*Rockefeller Travelling Fellowship.

Recently papers have appeared in the literature describing quite a large series of cases. Nearly all these are from the U.S.A. Munro (1934) describes 62, Kunkel and Dandy (1939) 48, Dyke and Davidoff (1938) 24, and Munro and Merritt (1936) reviewed the surgical pathology of 105 confirmed cases. But the largest series of all was that of Allen, Daly and Moore (1935), who described 245 cases out of a material of 3,100 autopsies on patients from mental hospitals. This represents 7.9%. Holt and Pearson (1937) describe three cases from mental hospitals which were diagnosed by encephalography. Two of these three cases were cured both mentally and physically, and the third developed an osteomyelitis of the finger which ultimately led to his death. These three cases all had bilateral hæmatomas.

Munro, an acknowledged expert on head injuries, states that it is regrettable that when car accidents are becoming increasingly more common and thus increasing the toll of head injuries, the lesion is not more frequently diagnosed. The figures of Allen, Daly and Moore, make it quite clear that mental patients should be investigated more carefully with subdural hæmatoma in mind.

*Etiology.*—Subdural hæmatoma is far more common in men than in women. Various ratios have been given by different authors, but all are agreed as to the great predominance in men. The condition may occur at any age from intra-uterine life upwards. In a large percentage of cases there is a history of trauma, either recent or remote. But most observers are agreed that a not inconsiderable proportion of the cases give no history of trauma at all. In Kunkel and Dandy's series 39.6% gave no history of trauma and only one out of the 48 cases showed a fracture of the skull. None of Dyke and Davidoff's showed fracture. In the present series of 30 cases (less four children to be described elsewhere) 11 of the remaining 26, or 42.3%, gave no history of trauma, and in no case was there evidence of a fracture of the skull on X-ray. Even allowing for the fact that many such patients are poor witnesses, for a number of them (9 out of 26) showed some mental changes, it is likely, considering also the absence of fracture of the skull, that any gross trauma, sufficient for the patient to remember, is not an essential factor in the ætiology.

Leary (1934) stresses the importance of alcoholism, which he found a common feature in his cases. He states that it probably acts in two ways:—

(1) By producing an œdema of the arachnoid, and thus serving mechanically to favour rupture of bridging veins, and (2) alcoholics are more addicted to injury than normal individuals.

Leary also found significant subdural hæmatomas in 10% of a series of several hundred fatal traumatic cases.

*Pathology.*—It is generally accepted that the condition is initiated by a tearing or rupture of the small veins traversing from the arachnoid to the dura. This laceration in more than half the cases follows trauma. In the remaining cases there may be a predisposing pathological lesion of the veins. In some cases it is possible that a gross rise in venous pressure, relative to the pressure in the subdural space, would allow a spontaneous rupture in an otherwise healthy vein or venule. But at the moment this must remain a conjecture. The bleeding presumably continues until the venous pressure is overcome by the general subdural pressure. As with hæmatomas elsewhere in the body a reaction of fibroblasts occurs at the periphery, and a capsule is ultimately formed. The blood may or may not remain fluid, and later, in rare cases, the hæmatoma calcifies.

Dyke and Davidoff maintain that a space exists between the hæmatoma and the dura, on the one hand, and the arachnoid on the other. This, they say, is a pathognomonic radiological sign. We find this view difficult to accept owing to the expanding nature of the process, and the somewhat raised intracranial pressure usually associated. Moreover, we have not been able to find the sign in any of the present series of cases. The situation of the hæmatoma is nearly always on the lateral convexity of the brain, the medial spread being limited by the falx. Why

this is the site of predilection is as yet unexplained. Possibly it is due to the greater number of veins in that situation which are draining to the superior sagittal sinus. Bilateral cases are not uncommon.

The clinical picture, in common with that of most other expanding processes in the cranium, varies very much. At one end of the scale are those cases which present only mental changes and headache. At the other end are cases with symptoms and signs of gross intracranial pressure. Frequently there are no localizing signs, and even if the correct diagnosis is made clinically, often there is nothing to indicate on which side the lesion is situated.

#### Material

Table I consists of 30 cases, four of which are dealt with in another paper and are of the infantile type. All the cases are collected from Professor Olivecrona's neurosurgical clinic in Stockholm. This table shows the years during which the cases

TABLE I.

Year	Number of subdural hematomas	Total expanding processes	Percentage subdural hematomas
1931	1	89	1.2
1932	1	106	0.9
1933	0	156	0.0
1934	2	172	1.2
1935	2	219	0.9
1936	3	197	1.5
1937	8	226	3.5
1938	7	242	2.9
1939 (to June)	6		

were admitted. Since 1937 these cases represent about 3% of all intracranial expanding processes admitted under Professor Olivecrona, and in 1939 the percentage is likely to be even higher (six cases in the first half of the year).

Table II (pp. 4, 5, 6 and 7) which is after Kunkel and Dandy, but slightly modified, classifies the chief features. It will be noticed that the cases are all males. The oldest was 67 years and the youngest 17 years (two cases). The average age was 44 years, which approximates closely to Kunkel and Dandy's figures (41.2 years), and Dyke and Davidoff's (44.5 years).

Headache was present in every case and it was always the first cerebral symptom. Of those 15 cases in which a history of trauma was elicited, a latent interval occurred in seven (43%). This varied from a few days to three months, and in one case twenty-one years. In such a case it is of course doubtful as to whether the injury was associated with the hæmatoma. In nine cases there was obvious mental confusion, 10 cases suffered from nausea and vomiting, 12 from diplopia, 20 had some degree of papilloedema. Papilloedema was by far the most constant sign. Table II shows how physical examination was almost entirely negative in other respects, and it was usually impossible to localize the side of the lesion.

A confident clinical diagnosis was made in seven cases, and in a further four cases it was put forward as being rather more likely than tumour. In the remaining 15 cases cerebral tumour was diagnosed. It is hoped to show later that radiology leads to a far more accurate diagnosis not only of localization, but also of pathology.

Twelve of the hæmatomas were on the right and twelve on the left, and two were bilateral. Of these cases one was in the left temporal region and one around the pituitary.

Fourteen cases were discharged cured, one of which (No. 1) died five years later of a bronchial carcinoma. (This represents a 54% cure rate.) Three were definitely improved. Five were not fit for any work, and one of these developed a right

TABLE II.—SUMMARY REPORT OF

Patient, No. and date of admission	Age, sex, (all male)	Chief symptoms	Type of trauma before admission	Duration of symptoms	Latent interval	Headache	Nausea or vomiting	Drowsiness	Diplopia	Vertigo	Convulsions	Coma	Mental confusion	Papilloedema	Involvement of other cranial nerves	Motor and sensory changes
1 E. T. 59/31 12.1.31	37	Headache 8 years on and off. Un- conscious once 3 weeks before admission	Nil	8 yr.	Nil	+	-	-	-	-	-	-	-	+	R.V. VII	Nil
2 G. J. 2538/32 10.10.32	17	Headache fol- lowed injury	4 months before admission hit on head by piece of iron	4 mo.	Nil	+	+	-	+	-	+	-	-	+	Nil	Some rigidity of neck
3 K. J. 1679/34 18.6.34	19	Headache 2 months	Nil	2 mo.	Nil	+	+	+	+	-	-	-	-	+	L. VI	Nil
4 L. W. 3191/34 12.11.34	37	Headache	5 months before admission fell on head off bicycle	2 mo.	3 mo.	+	-	+	-	-	-	-	+	-	Nil	Weakness L. arm, L. leg
5 H. P. 1501/35 31.5.35	27	Headache 3 weeks	Nil	3 wk.	Nil	+	-	-	+	-	-	-	-	+	Right ptosis, R. III	Nil
6 P. P. 196/36 2.4.36	50	Headache	4 weeks ago fell on icy steps. No head injury	3 wk.	Few days	+	-	+	-	-	-	-	+	-	Nil	Nil
7 E. A. 269/36 12.5.36	52	Headache	2 months ago fell off bicycle on head	2 mo.	Nil	+	+	+	+	+	-	-	-	+	R. VII	Nil
8 K. P. 404/36 18.7.36	59	Headache 2 weeks (complicated by pituitary tumour)	2 weeks ago hit head	2 wk.	Nil	+	-	-	+	-	-	-	-	-	Nil	Nil
9 P. L. 186/37 1.4.37	60	Headache 4 months on and off	Nil	4 mo.	Nil	+	-	-	+	-	-	-	+	-	L. VI	Muscle power lessened
10 J. N. 322/37 4.6.37	55	Headache 4 weeks after injury	8 weeks ago head injury in car	4 wk.	4 wk.	+	-	-	-	+	+	-	+	+	Nil	Nil
11 A. H. 400/37 9.7.37	17	Headache 1 month	Nil	4 wk.	Nil	+	-	-	+	-	-	-	-	+	R. VI	Nil
12 J. H. 458/37 10.8.37	61	Headache 3 months after accident	4 months ago motor-cycle accident: head injury	1 mo.	3 mo.	+	-	-	+	-	-	-	+	+	Nil	Nil
13 K. D. 619/37 27.10.37	26	Mental changes, headache 4 months	Nil	4 mo.	Nil	+	+	+	+	-	-	-	+	+	Nil	Nil
14 G. G. 658/37 16.11.37	28	Headache 1 month	Nil	1 mo.	Nil	+	+	-	-	-	-	-	-	+	Nil	Nil
15 K. A. 740/37 27.12.37	40	Headache 3 years	Nil	3 yr.	Nil	+	+	-	-	+	-	-	-	+	Nil	Nil

## OF 26 CASES OF SUBDURAL HÆMATOMA.

Deep reflexes	Clinical Impression T.C. = tumour cerebri, S.D.H. = subdural hæmatoma	Straight X-ray findings	V. = Ventriculography, E. = Encephalography, Diagnosis on burring	Arteriography	Side of lesion	Recurrence after operation	Result	Follow-up
Normal	T.C. Glioma	Nil	V.	No	L.	No	Cured	Died 25.1.36 carcinoma bronchus. Liver and spine metastases
Normal	T.C. brain-stem	Nil	V.	No	L.	No	Cured	Last communication 24.11.36. Very fit
Normal	T.C.	Nil	V.	No	L.	No	Better: slight speech difficulty	28.1.36: Quite well
Left more active than right	Right S.D.H. or T.C. frontal	Pineal calcified normal place	E.	No	R.	No	Cured	7.4.39: Quite well
Normal	T.C. or S.D.H.	Pineal calcified $\frac{1}{4}$ cm. to left	E.	No	R.	No	Cured	14.4.39: Quite well
Bilateral Babinski	S.D.H.	Nil	E. No air in ventricles. Burring	No	L.	No	Cured	8.9.38: Quite well
Right Babinski	Left S.D.H. or T.C. frontal	Pineal calcified normal place	E. No air in vent. Hæmatoma found on burring	No	L.	No	Cured	1.9.37: Quite well
Normal	Pituitary adenoma chromophobe	Enlarged sella	Nil	Yes	R.	No	Improved. Still headache	X-ray treatment. Less well in 1939
Normal	S.D.H.	Nil	E.	No	Bilateral	Unconscious after operation. after (Extradural hæm. —reoperated)	Died 3 days after operation.	
Normal	T.C.	Nil	V. Only contra-lateral ventricle filled	No	L.	No	Cured	22.12.38: Quite well
Normal	T.C.	Not made	Burring	No	R.	No	Cured	31.3.39: Quite well
Normal	S.D.H. or T.C.	Nil	Burring	No	R.	No	23.11.38: Headache now and then; cannot work	
Normal	T.C.	Calcified pineal, normal place	Burring	No	Bilateral	No	Cared	17.4.39: Quite well
Normal	4th vent. tumour	Widened sutures. Dorsum sellæ thin	V.	No	L.	No	Cured	18.3.39: Quite well
Normal	T.C.	Nil	V.	No	R.	No	Operation, good result	29.9.38: In alcoholic home

TABLE II.—SUMMARY REPORT OF

Patient, No. and date of admission	Age, sex, (all male)	Chief symptoms	Type of trauma before admission	Duration of symptoms	Latent interval	Headache	Nausea or vomiting	Drowsiness	Diplopia	Vertigo	Convulsions	Coma	Mental confusion	Papilledema	Involvement of other cranial nerves	Motor and sensory changes
16 A. S. 96/38 5.2.38	39	Headache 4 months	5 months ago fell on back of head. Unconscious	4 mo.	1 mo.	+	+	-	+	-	-	-	-	+	Nil	Nil
17 E. P. 390/38 23.5.38	36	2 months epi- lepsy, 1 month headache	Head injury 3 months ago	3 mo.	1 mo.	+	-	-	-	-	+	+	-	+	Nil	Nil
18 J. E. 440/38 13.6.38	67	Headache 2 months	Nil	2 mo.	Nil	+	-	-	-	-	-	-	-	+	Nil	Nil
19 J. A. 490/38 4.7.38	47	Headache fol- lowing injury	Hit head on post 10 days ago. Not unconscious	10 days	Nil	+	+	+	-	-	-	+	-	-	Nil	Nil
20 M. J. 938/38 16.12.38	65	4 months ago spots before eyes, then headache	Nil	4 mo.	Nil	+	-	-	-	-	-	-	-	+	Nil	Muscle weakness, left greater than right. Slight atrophy left side. Deep sensitivity impaired
21 O. L. 83/39 17.1.39	44	4 months head- ache, vomiting, drowsiness	4 months ago hit head against post. (Admitted this after operation)	4 mo.	Nil	+	+	+	-	-	-	-	+	+	Nil	Nil
22 F. E. 160/39 28.2.39	43	Headache and pain in face 21 years. Nervous 2 years	Bullet wound in head 21 years ago. ? type of injury	21 yr.	Nil	+	+	-	-	-	-	-	+	-	Nil	Nil
23 A. F. 205/39 15.3.39	62	Pain and weak- ness R. arm 8 years ago. Dizzi- ness 1 year. Head- ache 1 month	Nil	8 yr.	Nil	+	-	-	-	+	-	-	+	+	Nil	Nil
24 B. R. 286/39 22.4.39	51	Headache 2 months. Dim- ness of vision 1 month	Fell off a bus 2 months ago, unconscious	2 mo.	Nil	+	-	-	-	-	-	-	-	+	Nil	Nil
25 G. B. 299/39 26.4.39	59	Headache 1 month	6-7 years ago struck on head by hammer. No headache	1 mo.	? 6-7 yr.	+	-	-	+	-	-	-	-	+	Nil	Right hand, arm, leg, weak
26 G. G.	51	Headache 1 week	6 weeks ago thrown off a cart. Hit head — un- conscious	1 wk.	5 wk.	+	-	-	+	-	-	+	-	+	Nil	Nil

hemiplegia after operation (Case 21). One case was complicated by a pituitary adenoma and is still receiving deep X-ray treatment (No. 8). Three cases died as the result of operation (11.5% mortality). Case 26 had not left hospital but was an operative success.

#### Radiology

*Plain films.*—Of the 26 cases all but two (11 and 25) were examined by "straight" X-ray. Five survey views of the skull were taken in each case, as is the routine custom—postero-anterior, half-axial, axial, right and left lateral (Lysholm, 1931).

T OF 26 CASES OF SUBDURAL HÆMATOMA, *continued.*

Deep reflexes	Clinical Impression T.C. = tumour cerebri, S.D.H. = subdural hæmatoma	Straight X-ray findings	V. = Ventriculography, E. = Encephalography, Diagnosis on burring	Arteriography	Side of lesion	Recurrence after operation	Result	Follow-up
Normal	S.D.H.	Thin dorsum sellæ	E. No air in vent. Diagnosis on burring	No	R.	No	Operation, good result	16.2.39: Cannot work. Headache
Normal	S.D.H.	Calcified pineal, normal place	E.	No	L.	No	Nov. 1938 weak left arm—tired—poor memory. Readmission March 1939, wasting L. arm, slight papillædema. Refused enceph.	Discharged
Normal	T.C.	Calcified pineal, normal place	Burring	No	R.	No	Cured	
Normal	S.D.H.	Calcified pineal, normal place	E.	No	R.	Drowsy following opn. Reoperated. No bleeding found	Died pneumonia 4 days after operation	
Right Babinski	T.C.	Pineal calcified, 5 mm. to left	V.	No	R.	No	Operation, good result	22.4.39: Feels weak, left hand stiff. Cannot work
Right Achilles ++	T.C. malignant glioma	Nil	V.	Yes	L.	No	Drowsiness less but right hemiparesis	
Normal	T.C.	Nil	V.	No	L.	Reoperated for extradural bleeding	Died 12.3.39. Uræmia	
Normal	T.C.	Choroid plexus calcified. No displacement	E.	No	R.	No	4.5.39: Discharged much improved	
Normal	S.D.H.	Nil	E.	No	R.	No	13.5.39: Discharged cured	
Normal	T.C.	Not made	V.	Yes	L.	No	11.5.39: Discharged cured	
Normal	S.D.H.	Nil	E.	No	L.	No	Operation successful. Not followed up	

*Findings on Plain Films*

- (1) No fracture was seen in any of the cases.
- (2) Evidence of increased intracranial pressure was found in only two cases:—
  - (a) No. 14—widening of sutures of vault.
  - (b) No. 16—thinning of dorsum sellæ.
 Increased digitations of the bones of the vault were not observed in any of the cases.
- (3) No gross bone changes or abnormal vascularization of the bones was seen.

(4) The pineal gland was seen to be calcified in eight cases, but only in two cases (Nos. 5, 20) was it displaced laterally.

(5) The choroid plexus was calcified in one case but was not displaced.

(6) Calcification of the hæmatoma was not seen. (Had it been present it would have been pathognomonic—see infantile type.)

(7) Theoretically the shadow of the skull on the affected side should be denser than on the normal side, even in the absence of calcification; for blood contains far more iron than brain tissue and iron is, of course, an element with a high X-ray absorption coefficient. However, I was unable to demonstrate any such finding.

*Pneumo-encephalography.*—At the Royal Serafimer Hospital encephalography rather than ventriculography is regarded as the method of choice in investigating the brain and intracranial contents, provided that there is no evidence of greatly increased intracranial pressure or a suspicion of a posterior fossa lesion. It has the obvious general advantage of giving information about the convexity of the brain, the basal cisterns, and occasionally the subdural space, as well as the ventricular system. Ventriculography in most cases only gives information about the ventricles. Air injection for encephalography is always performed by suboccipital puncture. Sjöquist (1937) described the method as follows: The patient is placed in the sitting position and the head is supported in front by an assistant. The operator sits behind facing the back of the patient's head. The suboccipital region is shaved and sterilized. The skin and deeper structures are anæsthetized with a suitable local anæsthetic. After puncturing the dura the needle is advanced about 2 mm. Using a 10 c.c. syringe 10 c.c. of liquor are removed by suction and an equivalent quantity of air replaced. This technique is repeated until about 30 c.c. of air have been injected. The patient is then sent to the X-ray department on a stretcher trolley.

This method is found to be more satisfactory than lumbar air injection. First, less fluid needs to be removed, for the spinal theca need not be drained and less air need be injected. Secondly, as a result of this the air does not travel up the spinal cord, and thus the cord is not irritated. The symptoms are found to be less irksome to the patient, presumably for this reason. In fact, many patients have no discomfort at all. At the Royal Serafimer Hospital this technique has been employed by the neurologists, neurosurgeons, and psychiatrists for the past ten years, and many thousands of cases have been investigated by this means. Not only has there been not a single fatality, but in expert hands the ventricles have been satisfactorily filled with air in over 90% of cases.

In this material 11 cases were examined by encephalography. Seven of these showed satisfactory filling of the ventricles, and the position of the expanding process could be located (see diagrams).

One of these seven cases proved later to be bilateral (see below). One case showed filling of the contralateral ventricle only. In this case (No. 19) sufficient evidence was obtained to justify a burr hole being made on the opposite side in order to introduce air. On burring the hæmatoma was found. In the remaining three cases no air was seen in the ventricles and ventriculography was next performed, as is the usual custom.

*Ventriculography.*—It will be noticed that ventriculography was employed more often than encephalography, but even so encephalography has been used relatively more often recently.

In investigating subdural hæmatomas ventriculography has a special advantage in that on making the burr-holes and exposing the dura one may make a certain diagnosis of hæmatoma from the appearances of the dura, and thus air need not be injected at all.

Sixteen cases were examined by this method, including two of the three cases above. The other case was explored on clinical grounds alone. This accounts for all but one of the cases (No. 8), where a pituitary tumour was present, and air examination of the brain was not regarded as being indicated. The subdural

haematoma in this case was found by chance, and it was situated in an atypical position (*see later*).

Of these 16 cases six were diagnosed on making the burr holes. Of the remaining 10, satisfactory filling of the lateral ventricles was obtained in nine cases (*see diagrams*). In the tenth case (No. 10) air was present only in the contralateral ventricle. As is customary in such cases a frontal burr hole was made on the affected side in the hope that air could be injected by this means. However, on burring, the dura was found to show the typical appearances of an underlying haematoma, and so the air injection was not pursued.

The air-filled encephalograms and ventriculograms will now be considered together, for they show common features. To summarize the material we have:—

Clinical diagnosis .. .. .	1
Chance finding with pituitary .. .. .	1
Ventriculogram burr-hole diagnosis .. .. .	6
<i>Encephalogram—</i>	
Good filling .. .. .	7
Contralateral ventricle filling .. .. .	1
(Non-filling .. 3)	
<i>Ventriculogram—</i>	
Good filling .. .. .	9
Contralateral ventricle filling .. .. .	1
Total	26

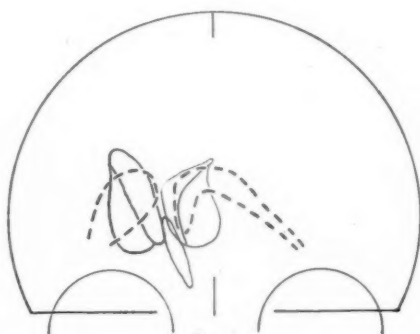


Fig. 1.  
CASE 2.

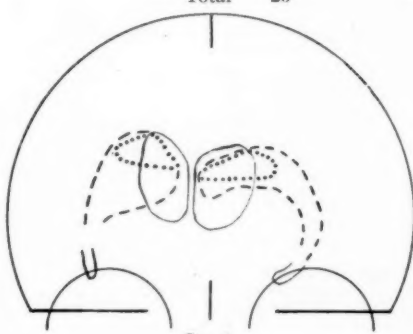


Fig. 2.  
CASE 3.

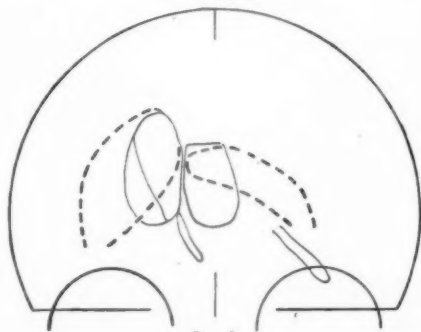


Fig. 3.  
CASE 4.

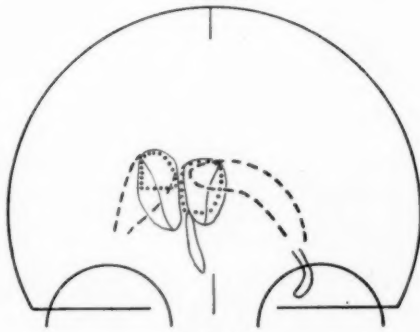


Fig. 4.  
CASE 5.

———— Pars anterior (patient supine).  
 ..... Cella media (patient sitting).  
 - - - - - Pars posterior (trigonum) (patient prone).

Of the 18 air-filled cases, 12 showed remarkable similarity (Nos. 2, 3, 4, 5, 14, 15, 20, 21, 23, 24, 25, and 26). Drawings were made of the ventricular systems in all these cases, and for the most part, except for the demonstration of falx pressure, the anteroposterior drawings are the most important. Reduced reproductions of these drawings are shown (figs. 1-12). For the sake of simplicity and diagrammatic

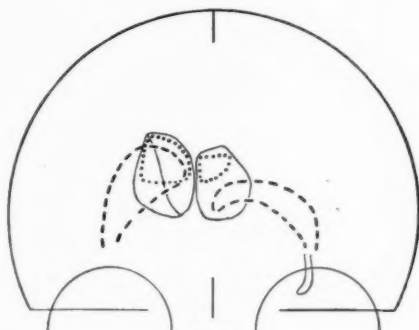


Fig. 5.  
CASE 14.

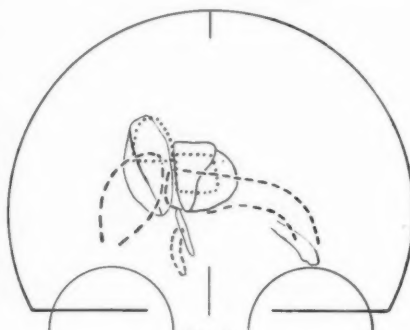


Fig. 6  
CASE 15.

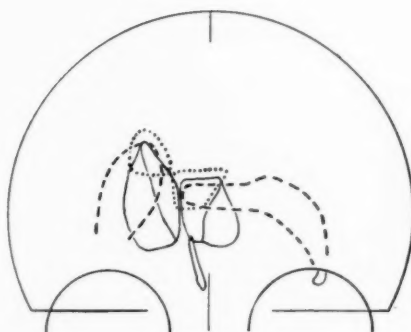


Fig. 7.  
CASE 20.

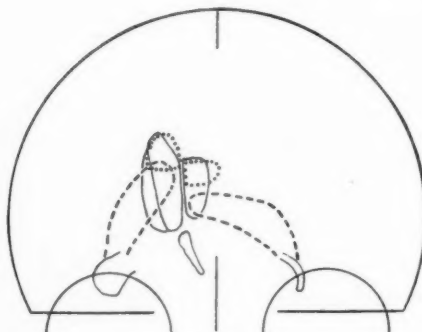


Fig. 8.  
CASE 21.

— Pars anterior (patient supine).  
..... Cella media (patient sitting).  
- - - - Pars posterior (trigone) (patient prone).

representation the lesion is represented on the same side in each case. (Fig. 9, Case 23, showed a congenital anomaly—a cyst of the septum pellucidum.)

The features noted are :—

#### A. Anterior Horns.

- (1) Marked septum pellucidum shift, with negligible angulation.
- (2) Shift, and angulation of the anterior part of the third ventricle with the sagittal plane.
- (3) Angle between septum pellucidum and third ventricle.

- (4) Marked dilatation of contralateral ventricle.
- (5) Very slight or absent dilatation of lateral ventricle on affected side.
- (6) Flattening of roof of lateral ventricle on affected side.
- (7) Elevation of roof on contralateral side.
- (8) Normal position or various grades of medial displacement of temporal horn on affected side.

**B. Sitting Position (pars media filled).**

- (9) More marked flattening of roof of lateral ventricle on affected side.
- (10) Greater displacement of septum pellucidum.

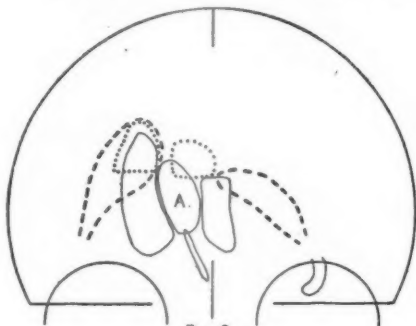


Fig. 9.

CASE 23.  
A, Septum pellucidum cyst.

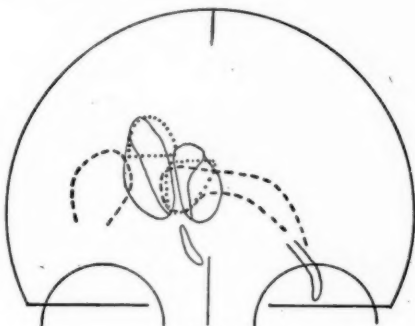


Fig. 10.

CASE 24.

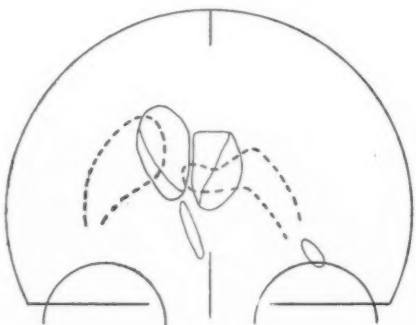


Fig. 11.

CASE 25.

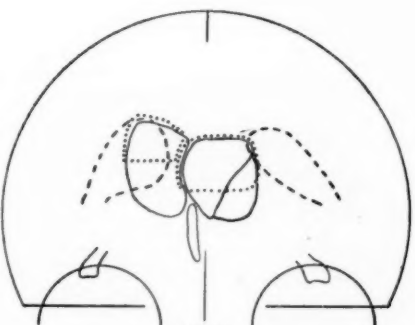


Fig. 12.

CASE 26.

- Pars anterior (patient supine).  
 ..... Cella media (patient sitting).  
 - - - - - Pars posterior (trigone) (patient prone).

**C. Trigone.**

- (11) Flattening of roof.
- (12) Slightly less shift of septum pellucidum than in B (pars media) but greater than A (pars anterior).

**Lateral View.**

Evidence of falx pressure in cases of great shift of septum pellucidum.

Superimposed drawings of these 12 cases were made, and it was found that they corresponded very closely. However, the result was so complicated in appearance that a synthetic drawing of the "average" was compounded in the three positions: A. Supine (pars anterior); B. Sitting (pars media); and C. Prone (pars posterior)

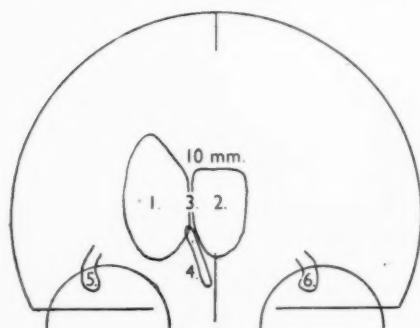


Fig. 13.

Mean drawing in supine position. Note: 10 mm. shift of septum. Medial displacement of 6.

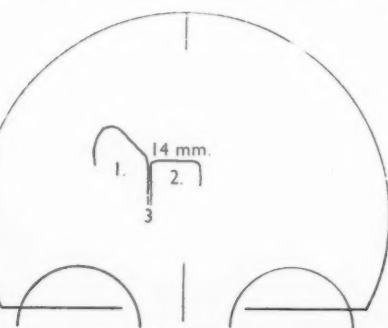


Fig. 14.

Mean drawing in sitting position. Note: 14 mm. shift of septum.

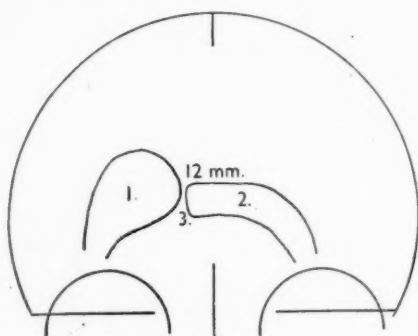


Fig. 15.

Mean drawing in prone position. Note: 12 mm. shift of septum.

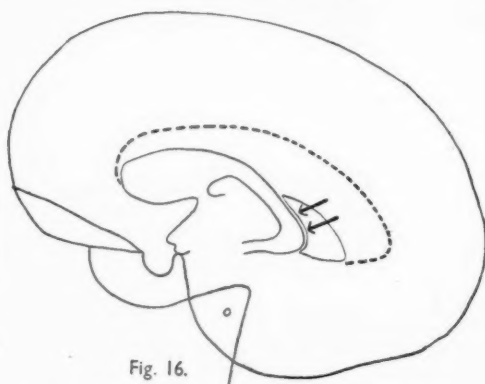


Fig. 16.

Typical lateral drawing (Case 20). Note: Falx pressure; contralateral ventricle dotted.

#### KEY TO FIGS. 13, 14 AND 15.

1. Contralateral ventricle: dilated, lateral roof high.
2. Homolateral ventricle: not dilated, roof flat.
3. Septum pellucidum: shifted but vertical.
4. Anterior part of third ventricle: upper part shifted laterally; forms angle with septum pellucidum.
5. Contralateral temporal horn: normal position.
6. Homolateral temporal horn: medial shift.

These drawings (figs. 13, 14, 15) show the above tabulated findings more clearly. The mean shift of the septum pellucidum in the three positions was measured and found to be 10 mm. anteriorly, 14 mm. in the cella media, and 12 mm. in the trigone region. One would expect this to be so with a laterally placed lens-shaped expanding

PLATE I.



FIG. 17.

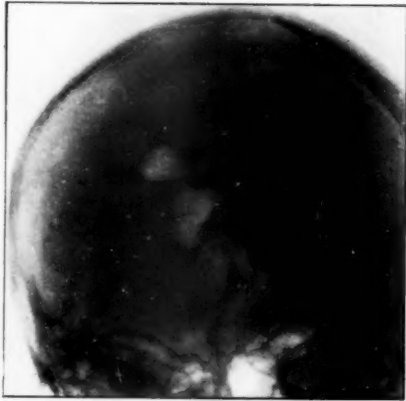


FIG. 18.

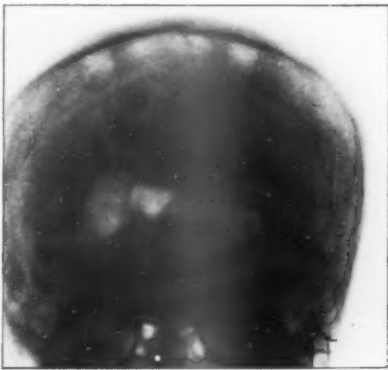


FIG. 19.

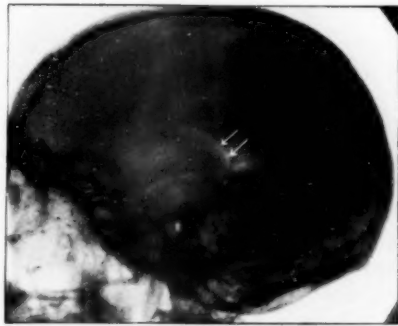


FIG. 20.—Arrows indicate falx pressure.

FIGS. 17-20 show radiographs taken in positions shown in figs. 13-16.

PLATE II.

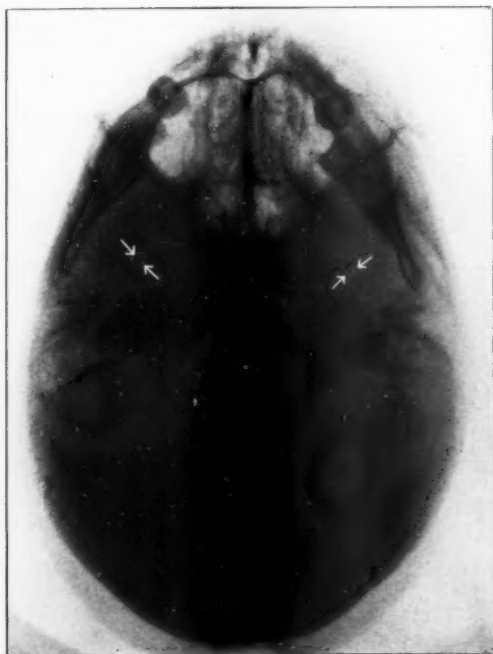


FIG. 21.—The foramina spinosa are circled.

process. Fig. 16 shows a typical lateral drawing. Figs. 17-20 show radiographs taken in positions shown in figs. 13-16.

The greater or lesser degree of the medial shift of the temporal horn depends upon the degree of spread of the hæmatoma into the temporal fossa.

This feature, in combination with the others, is regarded by us as being the

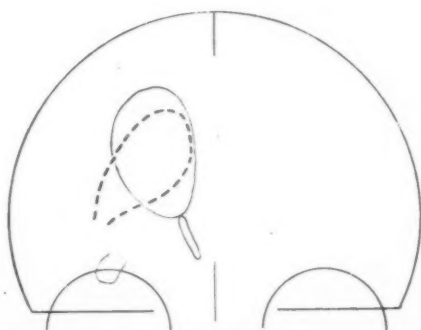


Fig. 22.

CASE 19.—Filling of contralateral and third ventricles only.

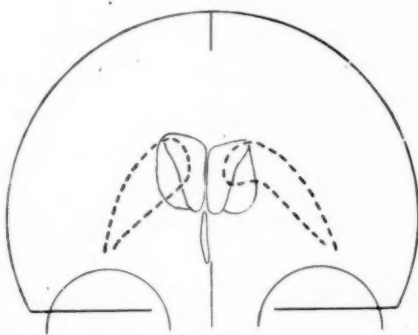


Fig. 23.

CASE 17.—Very slight lateral shift; no classical characteristics.

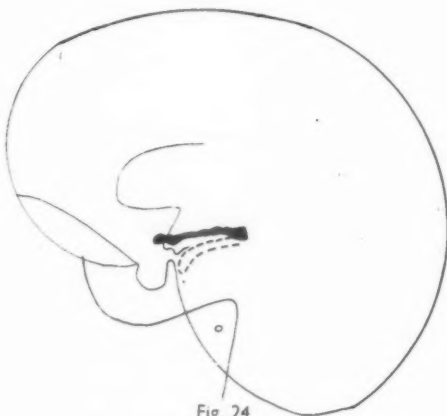


Fig. 24.

CASE 22.—Temporal horn in black; elevated and ragged. Normal situation dotted.

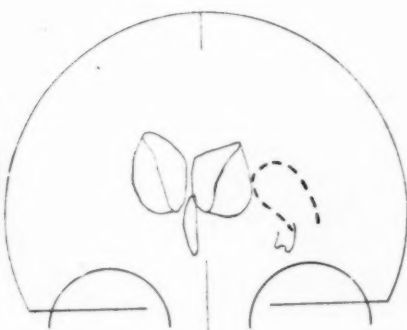


Fig. 25.

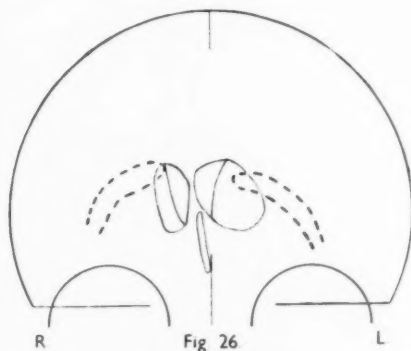
CASE 22.—Note medial and upward displacement of temporal horn.

most characteristic sign of the lesion. Although it cannot be called pathognomonic, for theoretically any expanding lesion of the same shape and position in the cranium could produce similar findings, in practice this must be very rare. Other expanding processes in this region will be the subject of a further research at the Röntgen

Department of the Royal Serafimer Hospital. But it is likely that a meningioma in the necessary situation is the only process which could simulate it, and had a meningioma grown so large it is highly likely that changes in the overlying bone or in the vessels would be present.

One such case was observed recently in which the pneumo-encephalographic features were typical of a subdural hæmatoma. There were no changes in the overlying skull, but the foramen spinosum on the affected side was much enlarged (fig. 21, Plate II). This indicated that the middle meningeal artery was hypertrophied, thus confirming the diagnosis of meningioma. An infiltrating growth such as a malignant glioma would be likely to produce localized deformities as well as general deformity, i.e. irregularity of contour of the lateral part of the affected ventricle, in contrast to the distant pressure effect of the subdural hæmatoma.

Of the remaining six cases which were air-filled, two (Nos. 10 and 19) showed filling of the contralateral and third ventricles only (fig. 22). These structures showed the features described above. Thus 14 out of 18 cases showed what I call the "classical picture".



CASE 9.—Bilateral hæmatoma. Note absence of classical characteristics. See text for description.

Of the remaining four cases, two (Nos. 1 and 17) showed almost normal appearances (fig. 23, Case 17), with just a slight displacement of the septum pellucidum. Both of these cases had small subdural hæmatomas.

One case (No. 22), with a hæmatoma in the base of the middle fossa, gave an atypical picture. Here a lateral drawing (fig. 24) is included to show the elevation and raggedness of the temporal horn. In the frontal view (fig. 25) the temporal horn is displaced medially and upwards. The ragged character of the temporal horn (lateral view) is difficult to explain, for it suggests an intracerebral lesion. This was in fact the radiological diagnosis, but at operation only a temporal subdural hæmatoma could be found. The patient died but no autopsy was obtainable.

One case (No. 9)<sup>1</sup> showed an atypical picture (fig. 26). This was not recognized at the time, and the condition was assumed to be merely a left-sided subdural hæmatoma—the diagnosis was made clinically and the encephalographic appearances seemed to confirm it. At operation a left-sided subdural hæmatoma was found. However, the patient did not recover consciousness after operation, repeated extradural hæmatomas formed and he was reoperated upon, but died, after three days, without recovering consciousness. At autopsy a subdural hæmatoma, 1 cm. thick,

<sup>1</sup> This case and Case 4 were described by Professor Ingvar and Dr. Ask-Upmark in their paper on subdural hæmatoma (*see* References).

was found on the other (right) side. Thus there are two bilateral cases in the series. In the other case the hæmatoma on each side was discovered on burring, preparatory to making a ventriculography. Bilateral hæmatoma should thus be borne in mind. On re-examining the encephalographic drawing of Case 9 in the light of the knowledge gained at autopsy one observes :—

(1) That neither temporal horn was filled. This might or might not have been of assistance, but if both were displaced medially, or if only the horn on the contralateral side was displaced medially, a bilateral lesion is certain. If the contralateral temporal horn was not displaced the diagnosis would be made more difficult.

(2) The drawing does not display some of the typical features shown in the 12 typical cases.

(a) There is no flattening of the roof of the "affected" lateral ventricle.

(b) There is no dilatation, but rather a diminution in size of the contralateral ventricle, and moreover the lateral border of its roof is not elevated but depressed.

(c) The third ventricle (anterior part) is practically vertical instead of being angulated.

In the light of these findings, and noting the contrasting appearances as compared with the typical cases, one should be very suspicious of the presence of a bilateral hæmatoma, in such a case.

*Pneumo-encephalographic technique.*—Lysholm's (1937) technique has been followed throughout. By utilizing his nine standard projections, and given sufficient air, it should be possible to outline the whole ventricular system and synthesize it by drawings. In practice this does not usually occur without special manipulation of the head, e.g. filling the temporal horn or fourth ventricle, and then taking subsequent pictures.

*Technique for subdural hæmatomas.*—Three pictures are taken in the supine position (Lysholm Ventriculogram Part I), and then examined. If a subdural hæmatoma, or in fact any expanding process in the lateral cerebral region, is suspected on the findings of these pictures, one then takes two pictures (one anteroposterior and one lateral) in the sitting position. By performing this manœuvre early in the examination a negligible quantity of air is lost from the ventricular system (the head is moved so that no flow of air through the aqueduct and exit foraminæ is allowed), and thus the cella media is examined.

The temporal horn of the affected side is next examined. It is very rarely filled on the first three pictures, so active manipulation of the head is necessary. This is quite simple—the head is merely turned so that the suspected temporal horn lies uppermost, and so air is allowed into it. The head is then replaced, the air being trapped in the temporal horn. The first three projections are then taken again. If the temporal horn has not been filled the process must be repeated. The contralateral temporal horn may then be filled by a similar manœuvre, as a control. This is only necessary in diagnosing very slight displacements of the temporal horn. The disadvantage of manipulating air into the contralateral horn is that some air is lost from the affected lateral ventricle, via the foramen of Monro, air which is required in examining the trigone of the affected side.

After examining the temporal horns one places the patient in the prone position and takes the remaining standard projections. It is very rarely necessary in investigating this particular lesion to take any more pictures.

*Falx pressure.*—Pressure by the falx on the trigonal region of the affected side is very commonly seen in these cases. It produces a quite characteristic deformity, and in the literature it is frequently interpreted as being due to pressure from an expanding process.

A reconsideration of the anatomy of the falx cerebri may assist in making the sign clearer. It will be remembered that the anterior part of the ventricle cannot come in contact with the falx at all, for the falx lies too high up and too far anteriorly.

Lateral pressure on the trigone, however, will cause it to come in contact with the falx almost immediately. It will then herniate under the falx and thus an impression is made on the trigonal outline (fig. 27). This sign is best seen in the projection VII (Lysholm) lateral projection—prone position, head not turned to right or left. If the posterior horns of the lateral ventricles are large, and if the quantity of air is not great, the air-fluid level may be too far posterior to see the falx impression. It is occasionally seen, however, with the head in the lateral position.

Fig. 28 is a reconstruction from a number of half-axial views of cases of subdural hæmatoma. It is semidiagrammatic and is intended further to clarify the condition. It is a view of the ventricular system from above in the classical type of subdural hæmatoma displacement. A cross section of the hæmatoma is shown on one side. The tentorium and the anterior borders of the middle fossæ are shown. The part of the falx which comes in contact with the ventricular system is shown, as well as the anterior portion. Note the slimness of the latter portion. The trigone is shown herniating under the falx. It also shows the relative dilatation of the contralateral ventricle, the curvature of the septum pellucidum, and the shift of the temporal horn on the affected side. In Case 2, when ventriculography was performed, some clear fluid was removed on puncturing the brain, and when the air was injected most of it was seen to be subdural, and outlining the falx (figs. 29, 30). This is the only case in which a complete outline of the falx has ever been observed at the Royal Serafimer Hospital.

*Stereoscopy.*—In the Lysholm technique stereoscopy is very rarely employed. Lysholm has found that by manipulating the head in various directions the whole ventricular system may be filled, and by taking pictures in two right-angled planes with the head in the various positions, localization of any deformity may be made. Half-axial views are also taken, for they provide an additional "dimension" and are of help occasionally in orientating deformities.

Finally, in every case a drawing is made of the ventricular system both in antero-posterior and lateral views. This provides a more accurate geographical plotting of the lesion than stereoscopy, which is subjective rather than objective.

*Arteriography.*—This is a useful ancillary method in the localization of intracranial expanding processes, and very often leads to a more accurate pathological-anatomical diagnosis than does pneumo-encephalography.

This investigation was performed on three of the cases. The first case (No. 8) was complicated by a pituitary tumour and only a lateral arteriogram and venogram were taken. No abnormality was discovered. In the other two cases (21 and 25) a malignant glioma was suspected on the history and clinical findings. The ventriculogram performed in each case showed the characteristic picture of a lateral expanding process (figs. 8, 11). Had the arteriogram shown an inoperable malignant glioma, the patient would have been saved an unnecessary operation.

Neither lateral arteriogram showed any abnormality, nor did the venograms.

The antero-posterior picture, however, in both cases showed a pathognomonic appearance both in the arteriograms and venograms (fig. 31). It will be seen that no contrast penetrated the hæmatoma, thus giving it a negative outline.

The arteriograms were taken towards the end of the injection of 8 c.c. of thorotrast—the venograms three seconds later. A stationary Lysholm grid was used incorporated in an apparatus designed by Lysholm-Schönander. This apparatus fits into the Lysholm-Schönander skull table and merely consists of a grid and a cassette holder for two cassettes, one above the other. The cassettes are separated by a tray of lead. The upper cassette and lead tray are quickly removed after taking the arteriogram. The second cassette is then in position for the taking of the venogram.

*Post-operative air filling.*—In two cases (Nos. 10, 13) air was injected into the

PLATE III.



FIG. 27.—Trigone ↓↓ Contralateral ventricle ↓ ↓. Note similarity to fig. 20.

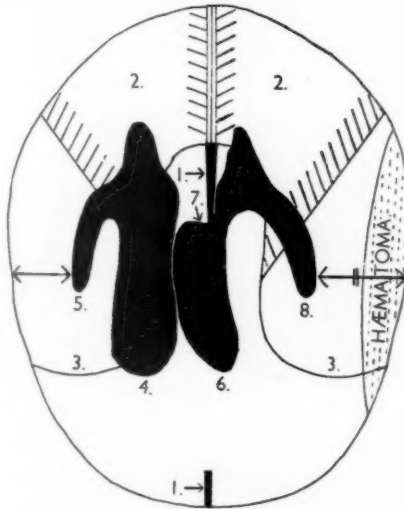


Fig. 28.

Semi-diagrammatic representation of ventricular system seen from above. Constructed from half-axial views.

Key.—1. Falx in cross section. 2. Tentorium. 3. Anterior border of middle fossa. 4. Contralateral ventricle. 5. Contralateral temporal horn (normal position). 6. Homolateral ventricle. 7. Region of falx pressure and herniation under falx. 8. Affected temporal horn—shifted medially. Note difference in length of  $\longleftrightarrow$  and  $\longleftrightarrow$

PLATE IV.



FIG. 29.—Air along falx and tentorium in subdural space.



FIG. 30.—Half-axial view of fig. 29.

PLATE V.



FIG. 31.—Arteriogram. Note absence of vessels at site of hæmatoma (right side).

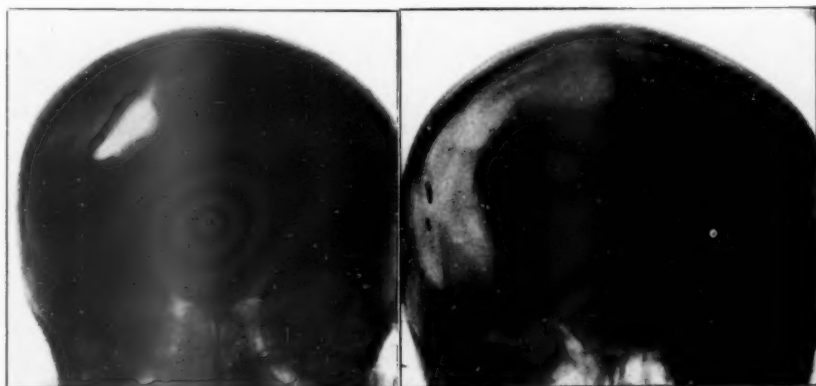
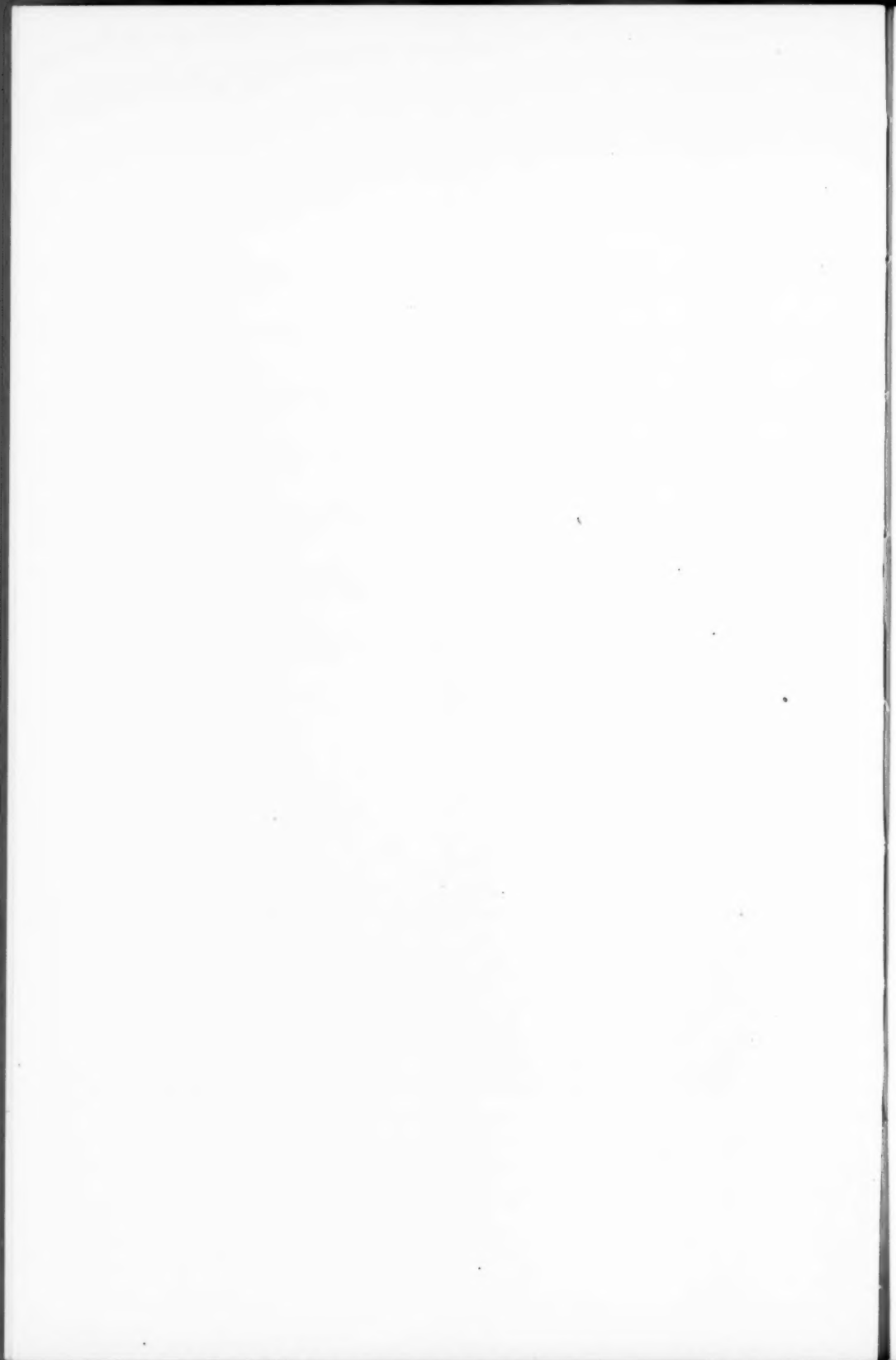


FIG. 32.—Air replacement after operation. Note fibrous strand traversing hæmatoma cavity.

FIG. 33.—Air replacement after operation (bilateral case). Note silver clips at sites of drainage.



space left after evacuation of the hæmatoma. This was performed about ten days after operation.

Case 10 (fig. 32) shows an unusual feature. Running across the middle of the hæmatoma is a fibrous strand.

Case 13 (fig. 33) was one of the two bilateral cases in the series. The radiogram illustrates very well the extremely marked reduction in brain volume which occurs in such cases. Perhaps one should rather call it "brain anæmia", for probably the brain itself is not compressible.

#### DISCUSSION

The question at issue is whether or not there are constant and characteristic radiological findings in chronic subdural hæmatoma, and if one is agreed that there are, whether or not they are pathognomonic. Let us consider the straight X-rays first. It is generally agreed that fracture of the skull is extremely rare. Dyke and Davidoff found no skull fracture, nor was there any in the present series, and Kunkel and Dandy found fracture in only one case.

In the present series evidence of increased cranial pressure as shown by bony changes in the skull could be found in only two cases. Dyke and Davidoff, on the other hand, found atrophy of the sella turcica in 11 of their cases (45.8%). But they virtually contradict this later in their paper in discussing the pneumo-encephalographic findings, they say: "The entire ventricular system shows a marked shift to the contralateral side. In spite of this, the accompanying roentgenographic and clinical signs of *generalized increased intracranial pressure are minimal or absent*" (italics mine).

Kunkel and Dandy's findings are far more in keeping with the findings in the present series. Only two cases showed evidence of increased pressure in the straight X-rays, and one of these was an infant aged 9 months, whose skull would of course show signs of increased pressure long before that of an adult.

The position of the pineal gland is of importance. In this series it was visualized in eight cases (30%) and was shifted laterally in two (25%) of these eight cases. Dyke and Davidoff saw it in 15 cases (62%) and it was shifted laterally in five (33.3%) of these cases. They claim that in 12 cases it was displaced posteriorly from 0.1 cm. to 1.1 cm. These latter figures are of very doubtful value, particularly as Dyke and Davidoff do not state what standards they adopt, what technique they use, or what they regard as the normal "limits" of the position of the pineal gland in different shaped skulls. It is difficult to understand how a posterior shift of 1 mm. can be diagnosed as being pathological when the gland itself may calcify to a diameter of 1 cm., and the skulls vary so much in shape that displacement, on the lateral view, of 1 mm. surely cannot be stated with any confidence to be a displacement at all. As the hæmatoma is nearly always laterally placed and at a considerable distance from the pineal gland, backward displacement must be of very doubtful significance. But they state: "... the pineal displacement was relatively great, particularly in comparison to the slight signs of increased intracranial pressure".

Lilja's (1934) statistical survey of 200 cases showing calcification of the pineal, which presented no radiological or clinical evidence of an intracranial expanding lesion, illustrates this point. The normal variation of position was quite considerable.

I consider that in subdural hæmatomas the only significant finding associated with the pineal gland is lateral displacement, i.e. that seen in A-P or P-A projection. Calcification of the hæmatoma would appear to be the only pathognomonic sign on the straight X-rays.

*Pneumo-encephalographic features.*—Let us first consider hydrocephalus. There is no unanimity of opinion on this point. Kunkel and Dandy state: "Hydrocephalus

of varying degree was present in six out of nine cases . . . the cause of hydrocephalus was compression of the aqueduct of Sylvius." Dyke and Davidoff, on the other hand, say: "The lateral ventricles were visible in 11 out of the 15 cases and their size was well within normal limits, in fact, towards the lower limits of normal, with the exception of one case."

These two views are completely opposed to one another. In the present series all cases showed some degree of hydrocephalus and as shown (in figs. 13, 14, 15) the average was quite considerable.

The cause of the hydrocephalus is not convincingly clear. Kunkel and Dandy state (*see above*) that it is due to compression of the aqueduct of Sylvius. Is this always so? If the aqueduct is compressed it must almost certainly be displaced also. What is the evidence for such displacement? The aqueduct, lying deep in the brain-stem, is below the pineal gland. If the pineal gland, which lies at the back of the third ventricle, is not displaced laterally, the aqueduct is still less likely to be displaced. When the pineal was calcified in Dyke and Davidoff's series only 33.3% of these cases showed lateral shift and in the present series only 25%. It is therefore by no means conclusive that the aqueduct is compressed. As Ingvar and Ask-Upmark (1938) point out, the brain-stem may be likened to the root of a vegetable, and this root is much more firmly anchored at the base of the skull by vessels and nerves than the rest of the brain. This makes it even more unlikely for the aqueduct to be displaced by a process impinging on the convexity of the cerebral hemisphere. The fact that the upper portion of the anterior part of the third ventricle is so constantly displaced does not alter the argument; furthermore the bottom of the third ventricle (*see* figs. 1-12) is not displaced. The aqueduct is deeper in the brain than the anterior part of the third ventricle, and further away from the expanding process, and has even less chance of being displaced.

The most convincing explanation of the hydrocephalus appears to be that of Munro (1938) who holds that when the subdural space is distended a certain number of arachnoid villi are put out of action. Thus the production-absorption balance of cerebrospinal fluid is upset and hydrocephalus results. But even this theory is not altogether satisfactory, for in the one bilateral case in the series with air filling (fig. 26) there is an absence of hydrocephalus. With so many more arachnoid villi pressed upon by the hæmorrhage one would expect an even greater hydrocephalus.

Kunkel and Dandy state: "In three cases (33.3%) the ventricle was entirely collapsed on the affected side." But frequently on performing a ventriculogram or encephalogram one finds that the affected ventricle is not filled. This applies not only to subdural hæmatomas but to a variety of expanding processes in this region. But if on finding such a state of affairs one makes a burr hole in the frontal region on the affected side one finds that invariably the affected ventricle can be filled. At the Royal Serafimer Hospital, out of thousands of X-ray examinations and autopsies, the affected lateral ventricle has never been seen to be completely collapsed.

Again Kunkel and Dandy state that: "The reduction in the ventricular volume may be greater in front, greater behind or essentially equal throughout." While admitting that the ventricular volume on the affected side is less than that on the contralateral side owing to the lateral and downward pressure, and the falx pressure, this difference in volume is not very great. Also it was found with only one exception (Case 26) that the anterior borders of the two lateral ventricles were side by side. The posterior border cannot be assessed, for in the normal the occipital horns on either side vary very much. In Case 26 the anterior border of the affected lateral ventricle was displaced less than 1 cm. backwards.

#### CONCLUSIONS

The only pathognomonic radiological sign on the plain X-rays—that of calcification—was never seen. It has been observed in children and one case will be recorded in

another paper by the author, but it seems likely that adults die if untreated before the lesion has time to calcify, a process which probably takes years to come about. The pneumo-encephalograms showed constant features (excluding the one bilateral and the atypical cases).

Thus although no finding can be said to be absolutely pathognomonic, the circumstantial evidence is so great that the operative approach should be made with this diagnosis in mind, and a diagnostic burr hole drilled. In this way the reflection of a large osteoplastic flap may be avoided and the patient is submitted to a minor rather than a major operation.

A more difficult problem is the bilateral case. As stated above in describing the encephalographic changes in the one case of this type with air filling, certain atypical features are noted, and should make one suspicious. Other ancillary aids should perhaps be sought. Arteriography would not help unless injection was performed on both sides. This routine is adopted in very few clinics. It is possible, however, that electro-encephalography might be of assistance.

Arteriography: In the antero-posterior position it gives a pathognomonic sign as described above, but it is very doubtful if this investigation is justified in subdural hæmatoma, for although injection of the internal carotid artery is a small operation in itself, thorotrast is probably not a harmless drug.

I wish to express my thanks to my chief, Dr. Erik Lysholm, but for whose inspiration and help this work would not have been performed. I also wish to thank Professor Herbert Olivecrona for kindly placing his material at my disposal, and for his advice.

Finally, my thanks are due to Dr. Erik Lindgren and all other colleagues at the Royal Serafimer Hospital for the kind assistance which they were ever prepared to give me.

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*Discussion.*—Dr. J. G. GREENFIELD said that Dr. Cumings and he had been investigating van den Bergh's reaction, as one of the ancillary methods of diagnosing subdural hæmatoma. Theoretically, if recent blood-clot was being absorbed and broken up, van den Bergh's reaction should be positive, and it was obtained in most cases of recent subdural hæmatoma.

A case in which bilateral calcified subdural membranes had formed presumably as the result of subdural hæmatoma, occurring during parturition or in infancy, was recently examined post mortem at the National Hospital, Queen Square. Fresh bleeding had occurred into the cavity between the calcified membrane and the skull on one side and led to the patient's death at the age of 16 years.

He agreed with Dr. Bull's summary of the pathology of subdural hæmatomata. It was difficult to understand why subdural hæmatomata continued to enlarge, sometimes, apparently, for months after the initial bleeding.

There were three theories for this continuous enlargement. Cushing and Putnam (*Arch. Surg.*, 1925, **11**, 329) said that the hæmatoma became covered with a membrane in which there were rather dilated thin-walled capillary vessels which continued to bleed into the substance of the hæmatoma for a long time under various provocations, with the consequence that fresh blood was always coming into the hæmatoma from this source. He was himself inclined to accept that view, because in every case examined fresh blood corpuscles were found in the hæmatoma, thus indicating recent hæmorrhage; and the only source from which it could come would be these dilated vessels.

The other two theories seemed more unlikely. One was that the broken-down blood in the hæmatoma, owing to its increase in osmotic pressure, absorbed cerebrospinal fluid or some other fluid from the neighbouring tissues. That would mean that eventually in the hæmatoma there would be a fluid containing probably low protein with a large amount of breakdown products of protein, which had not actually been found.

The third theory was that the hæmatoma was not really subdural at all but intradural, stripping off the so-called inner layer of the dura, which was very vascular, and therefore with a tendency to bleed. Those who had seen subdural hæmatomata either at operation or autopsy would agree that they could be stripped off the dura with the greatest ease, leaving a perfectly smooth dura behind. There was no evidence that the hæmatoma occurred in the layers of the dura, at least in the ordinary traumatic cases.

Major D. E. DENNY-BROWN said that the method of encephalography described by Dr. Bull sounded a very rational and successful procedure. It was remarkable that he got so little air in the subarachnoid space, and that presumably was why he had not seen the cleft appearance of subarachnoid air under the hæmatoma said to be characteristic of subdural hæmatoma by Dyke and Davidoff.

He had seen two calcified subdural hæmatomata. One of these was a case reported by Critchley and Meadows (*Proc. Roy. Soc. Med.*, 1932, **26**, 306 (Sect. Neur., 12)). The other case was in a woman aged 35 whose initial injury and onset of symptoms occurred at the age of 15. There were deformities of the skull like those which Dyke and Davidoff described in the sphenoidal fissure and the roof of the orbit, apparently only occurring as the result of the prolonged enlargement of the middle fossa by the subdural hæmatoma during the period of growth.

Perhaps he might add that he had come across a triple subdural hæmatoma. This was a patient in whom subdural hæmatoma was suspected, the usual burr holes were made, and bilateral subdural hæmatomata were drained with resulting recovery of consciousness. After about twelve hours deep stupor reappeared. Naturally it was suspected that there was obstruction of drainage, but investigation showed no such obstruction. The patient succumbed. At autopsy a very large occipital cyst, quite separated from the two parietal cysts by a partition, was shown to have been enlarged by a further collection of fresh blood. He was not aware of a description of a triple sac in the literature.

Dr. G. E. F. SUTTON said that Dr. Bull had mentioned another method of diagnosis, namely, the electro-encephalograph. He had recently seen a patient operated on by his colleague, Professor Rendle Short, on a diagnosis made by Professor Golla at the Burden Neurological Research Institute at Bristol. The diagnosis had been made by electro-encephalography, which showed, in contradistinction to the physical signs, exactly where it lay.

Mr. D. W. C. NORTHFIELD said he agreed that the clinical picture of subdural hæmatoma might vary widely, thus adding to the difficulty of making a correct diagnosis. The symptoms and signs might be only those of increased intracranial pressure due to a space-occupying lesion. The common problem was to decide between a malignant tumour, an abscess, and a subdural

haematoma, any of which might give rise to similar symptoms of a few months' duration and produce inconclusive localizing neurological signs or no such signs at all. On the other hand even outspoken focal neurological signs which would suggest a large intracerebral lesion might be produced by a haematoma. He described two cases illustrating this point.

At times the mode of onset and the distribution of the headache might be much more suggestive of subarachnoid than subdural bleeding, and the finding of a yellow cerebrospinal fluid would add to the difficulty of making a correct diagnosis. This was so in two other cases which Mr. Northfield described.

The other point he wished to raise in discussion was that of intracranial pressure. Headache was a constant complaint during some period of the history and was usually progressively severe and paroxysmal in type, denoting an expanding intracranial lesion. Although the haematoma might occupy a considerable amount of the intracranial space, yet objectively the intracranial pressure was seldom high and frequently abnormally low. In his own experience, papilloedema was not common. In only two cases of a recent series was the pressure of the cerebrospinal fluid abnormally high, and curiously enough in neither of these was papilloedema present. In one the lumbar pressure was 190 mm.; in the other the ventricular pressure was 600 mm., but the patient was congested from respiratory failure. In the remaining cases the pressure varied between zero and 150 mm. It would be argued that the lumbar pressures were not a true reading of the intracranial pressure owing to a partial block from herniation. This did not fully account for the findings because in some cases with a low lumbar puncture pressure, there was a good rise with jugular compression. The zero reading was of ventricular pressure, and in another case the ventricular pressure was such that fluid only trickled out slowly and a satisfactory measurement could not be obtained.

States of low intracranial pressure were frequently met with in cases of head injury, of long-standing hydrocephalus, and of tumours. It was perhaps most frequently seen in elderly persons with malignant tumours, and Dr. Greenfield suggested that this depended upon the greater roominess of the subarachnoid spaces which occurred in old age. Such an explanation was not applicable to the mechanics of all cases of subdural haematoma, for the low pressure was found in young as well as in old patients.

If W. J. Gardner's views (*Arch. Neur. and Psychiat.* (1932), 27, 847) on the factor of osmosis in subdural haematomata were correct they offered a possible explanation of the low-pressure phenomenon. The greatest transference of water should occur through the inner and thinner membrane, the surface of which was applied to the brain. Water would thus be abstracted from the brain which would become compressed *pari passu* with the expansion of the cystic collection; thus the total bulk of the brain and cyst might not be increased.

Dehydration might be partly responsible for the low pressure in a few very ill patients, but could not be the cause in the majority.

Professor JEFFERSON said that Lysholm's reputation was so great that nothing need be said about it to such an audience, but it was pleasant to feel that one of their colleagues who had been sitting at Lysholm's feet had come back imbued with his methods and spirit and had been able to give such a paper and to show such pictures as Dr. Bull had done that afternoon. There was considerable doubt in the radiological world as to the uniformity, or even the possibility, of obtaining appearances of subdural haematomata by encephalography or ventriculography, particularly the first method which they used so much at Stockholm. Only a few years ago, when von Storch and Munro (*New Eng. Journ. Med.* (1938), 218, 6) published a series of thirty-five cases they concluded that it was not possible to diagnose subdural haematoma by means of encephalography. This made Dr. Bull's present paper all the more important, because he had not only shown that it was almost uniformly possible to confirm the diagnosis in this way, but by a most interesting and careful dissection of the ventriculographic outlines he had shown, piece by piece, what was the meaning of the shift. He had made his case quite clearly that as a general principle only a tumour which was shaped like a haematoma and was situated where the haematoma was could fulfil all the requirements of the final picture.

Dr. J. W. D. BULL, in replying to Dr. Denny-Brown, said that an intra-hemispherical tumour such as a malignant glioma, would probably produce a localized deformity somewhere or other in the lateral wall of the affected lateral ventricle, without dislocation of the temporal horn.

He had been very interested in Dr. Denny-Brown's reference to the cases described by Dyke and Davidoff (1938) and two of his four infantile cases showed those appearances.

Dr. Bull thought that the disadvantage of using the electro-encephalogram only, was that it did not give the indication as to whether the lesion was extracerebral or intracerebral.

Mr. Northfield had raised the question of the clinical picture simulating a malignant glioma. It was for this reason that in Stockholm arteriograms were made on these occasions. Until they had realized the classical pneumo-encephalographic picture, it was the practice if a case was diagnosed as a malignant glioma, to make an arteriogram which gave the quite characteristic picture as described by Hemmingson (*Acta Radiologica* (1939), 20, 499). It was very important to be quite certain that the lesion was not a malignant glioma because, as Mr. Northfield had pointed out, so many of these cases might be cured.

## Section of Ophthalmology

President—MALCOLM HEPBURN, F.R.C.S.

[January 25, 1940]

### DISCUSSION ON GAS INJURIES TO THE EYE

**Mr. M. H. Whiting :** These remarks will apply only to eyes affected with mustard gas as I have no experience of other varieties.

*First use of mustard gas in the last war.*—Mustard gas was first employed by the German Army in the summer of 1917 about the middle of July. The gas attacks were severe and the British casualties were numerous. Between July 11 and 25 on the XV Corps front there were 2,350 cases. The 49th Division suffered so severely in five days that it had to be withdrawn from the line.

During 1917, 52,452 shell gas casualties were admitted to casualty clearing stations on the whole front, and 1,168 died in these units. To the hospitals on the lines of communications there were 40,566 admissions with 628 deaths. Thus about 10,000 were sent back direct from the casualty clearing stations to their units.

*Gas cases at Boulogne.*—The ophthalmic beds at Boulogne were in the 83rd General Hospital and in the summer of 1917 numbered 70; they were later increased to 120. They were under the general supervision of Sir William Lister, Ophthalmic Consultant to the Forces in France, and Mr. Goulden and I were the Medical Officers in charge.

The first gas cases we had were two or three men who had been exposed, without knowing it, to a gas attack. They were due for leave that day and on the way down to the Base the symptoms developed. On arrival at Boulogne photophobia and blepharospasm made them practically blind and they were sent up to our hospital. The same night we had a convoy of about 50 gas cases, and on the following days a succession of convoys. The majority of these cases were slight, and after a day or two in hospital were sent to convalescent camps from which they came up for observation.

*Degrees of affection.*—The cases could be divided into three classes :—

- (1) Slight cases with little or no corneal affection—about 75%.
- (2) Moderately affected eyes with slightly roughened cornea but not staining with fluorescein—about 15%.
- (3) Severe cases with the cornea definitely staining and corresponding conjunctival involvement.

Sir William Lister (History of the Great War, Medical Services, Surgery of the War, 2, 530) estimates that the first class were fit for duty in one to four weeks, the second class in four to six weeks, while many of the third class were evacuated to England and required variable longer periods to become fit for duty. There were also a very small number who had actual burns of the eyes and eyelids with liquid. Such cases followed the ordinary course of caustic burns of the eye.

*Clinical picture.*—All classes suffered from photophobia and blepharospasm. Only the third class complained of severe pain.

*Class I* had a moderate to acute injection of the exposed conjunctiva, the rest of the conjunctiva being relatively unaffected. The cornea was clear and bright.

*Class II.* The conjunctiva was white in the palpebral aperture and the rest of the conjunctiva was congested. The cornea was often slightly hazy but did not stain.

*Class III.* The conjunctiva was as in Class II but the changes were more pronounced and the cornea showed an orange-skin roughness and in many cases stained for a variable period.

*Pathological changes.*—The only eyes actually examined were those in severely affected cases in which death resulted from general causes. These showed a denudation of the corneal epithelium with a flattening of the remaining cells.

In Class III the white appearance of the conjunctiva in the early stage was due to coagulation and arrest of circulation in the conjunctival vessels. In the late stages the appearance was reversed, the exposed area being injected and the vessels sometimes remaining permanently enlarged, while the rest of the conjunctiva assumed its normal appearance.

In Class I the dilated vessels in the exposed area were due to direct irritation and the injection soon subsided.

*Treatment.*—At Boulogne and the other centres various lotions were used such as boracic, normal saline, weak eusol, and sodium bicarbonate. It was found that normal saline bathing with liquid paraffin drops was the most comfortable treatment and gave the best final results.

In the more severe cases with corneal affection, atropine drops once or twice a day made the patients more comfortable. Secondary infection in these was the most dangerous complication, leading to definite corneal ulcerations, and protargol and boracic lotion were employed. Possibly sparing use of mercury oxycyanide lotion would also be effective.

The changes produced by gas are almost instantaneous and I do not think that any measures directed to the chemical neutralization of gas are likely to help recovery and might even cause greater irritation. The treatment must be directed to the results of exposure to gas.

Shades or dark glasses were useful when the photophobia was considerable, but they should not be continued too long as there is a definite psychological element in many cases in the later stages.

*Results of gas affections of the eye.*—A large proportion of gassed cases have practically no corneal involvement and recover completely in from two or three weeks.

Some of the cases in the last war did not have to come as far as the base hospitals in France. Owing to the pressure on the bed accommodation in France, patients who required in-patient treatment for more than a few days had to be evacuated to England and progress there depended, no doubt, on the nature of the treatment which they received. Irritating treatment over a long period with antiseptics would certainly retard recovery and the use of cocaine should be forbidden.

On the other hand, bathing with non-irritating lotions might be required for some time, as there was often a slight persistent conjunctivitis.

The causes of late corneal degeneration which have been recently observed, though they must form only a minute fractional percentage of the total number affected with gas, are of interest. It is probable that some of these are the result of the actual injury with gas at the time, but others may be the result of improper treatment in the later stages.

At Boulogne, where over a third of all the gas cases were treated, only two cases of hypopyon ulcer occurred, and I can only recall two or three cases in which the eye was lost. These were said to be the result of actual liquid reaching the eye.

*Gas and the civilian population.*—The civilian population in England is now much better prepared to meet gas attacks than the Army was in France in 1917. Gas masks with protection for the eyes are universal, gas detectors are placed in all the populous districts, and people have been well instructed in the facts about gas attacks from the air.

**Mr. R. E. Bickerton** said his own experience of mustard gas did not date until after the war. He referred in detail to one mustard gas case which he had lately seen. This was the case of a regimental medical officer who had worked in a dugout which became soaked with gas after an attack. It was a place roofed over with corrugated iron and braziers had been put in. He and several orderlies were affected in the same way. They did not realize that anything was wrong until their eyes began to smart, and afterwards they had all to be led out of the dressing station and this man was sent down to the base. He got an attack of double pneumonia from which he nearly died. His eyes were particularly bad. Every form of treatment was applied, but nothing seemed to do much good. He recovered from his pneumonia after four months, his eyes remaining affected during the whole time, and afterwards it was discovered that he was subject to rises of temperature and had developed tuberculosis. That was not an unusual happening after mustard gas attacks. The speaker did not see this man until 1935, when it had become quite impossible for him to carry on his practice in London. The whole of his corneal epithelium had exfoliated and left Bowman's membrane exposed. A striking appearance was present around each eye in the form of two very white arcus rings. When his cornea began to go this arcus began slowly to excavate and left a deep groove all round the cornea. The arcus completely disappeared. The present result was that his left cornea was completely opaque. He had multiple corneal ulcers, and the speaker dare not use the cautery more than he could possibly help. He did on one occasion use carbolic acid, which rather soothed him, but he fancied it made the ulceration worse.

Turning to gases used in the last war, he was out at the Front during the first two gas attacks, the second of which took place in Ypres. The use of gas in any form was a very double-edged weapon, *vide* Armentières. He believed that the actual number of gas cases in the British Army was 150,000, and one in forty of those cases died. Some of the more severely gassed cases were still going on, and the eyes were disintegrating.

The first gas used was chlorine, bromine, and prussic acid. This was in the spring of 1915. It was difficult in an attack by such a gas either to breathe or to speak. Later they developed a cough which lasted for many months. They were unable to obtain a night's sleep on account of the irritation which began as soon as they lay down. But this gas did not very much affect the eyes; it caused oedema of the lungs. The men were brought in in an unconscious state, making breathing sounds like those heard on the filling up of a bottle. It was necessary to hold them up by their legs and squeeze out their chests; their lungs were simply water-logged. Most of the chlorine gas caused severe lung trouble. The only remedy at hand was atropine, and on the principle that atropine dried up the secretions to some extent it had probably some effect in such a condition. Later they were given atropine and morphine and their breathing became less heavy. They all got conjunctivitis to start with, but this very soon cleared up. He did not think boracic acid or any watery solution was of very much value in chlorine gas cases. The sanitary people

sent up vacuum glass flasks with rubber stoppers to collect samples of the gas, which were opened and closed in the gas cloud and that was how they discovered first of all that a mixed chlorine and bromine gas was being used, and also in addition to chlorine and bromine a touch of prussic acid. The prussic acid soothed the eyes rather than hurt them after the first whiff. But undoubtedly the gas most dangerous to life was phosgene. The men affected took about three hours to walk a mile and had to lie down at very short intervals on the way. Many cases of phosgene gas poisoning were brought in dead on the stretcher, the exertion of getting to and on the stretcher having simply stopped the heart. But phosgene was practically without any effect on the eyes, either immediate or remote, while so far as chlorine was concerned, though it irritated the eyes to some extent, most of the effect was on the lungs.

Mustard gas had the most destructive effect on the conjunctiva, cornea, and lower lids, also upon moist areas of the skin and the lining of air cells in the lungs. The whole of the front of the eye in the lower third became so opaque that the patient could not see through it. After a time the cornea began to bulge and perforated, first in one eye and then in the other. The first effect of the irritant in mustard gas cases was considerable watering, and the tears made a weaker solution of the gas, which weaker solution was rather worse than a strong one would be. Very few cases were gassed with fluid splashes, but usually the gas, owing to the tears, became more or less a gas solution not eroding itself in quite so quickly, but soaking into the corneal epithelium, getting down to Bowman's membrane, which rapidly softened and offered but little protection to the lamellæ of the cornea, between the layers of which the gas solution percolated. As a rule the irritating effect went on for years. Definite keratitis took place, ending in keratomalacia. In bad cases there was likely to be an iridocyclitis, and the irritation was at first considerable. After a time it became less painful but there was ulceration in the cornea, causing at almost regular intervals much trouble and discomfort. Abscesses occurred in the cornea which sometimes burst both ways into anterior chamber and conjunctival sac, and there was a recurring ulceration for years. The attempts to counteract it by watery lotions did not do much good. Oily solutions had much more effect, and if drops were used these should be in oil, and in castor oil rather than paroline. A very good solution was homatropine and cocaine in castor oil.

The question arose as to the removal of the eye on its becoming practically useless. He had never done that until the eyeball was actually perforated, and in such cases removal was a necessity. A good deal was said about stitching up the lids. In these old cases he thought that was one of the worst things to do, for the eroding process still went on and continued more rapidly if the lids were closed up altogether so that the beneficial effect of the air could not reach the eye. In a word, these very bad cases behaved in very much the same way as the cases in which the eye had been injured by vitriol throwing or a splash of caustic soda. Mustard gas was very similar, indeed, to vitriol in its action. The only certain remedy was to neutralize the gas from the beginning if possible. The only known antidote to the mustard gas group was chlorine, and a solution of sodium hypochlorite (1 : 800 or 1 : 1,000), used immediately as a lotion, might mitigate the severity of the damage or stop it altogether. The solution was slightly painful, but it did start the neutralizing action almost at once. In some cases phenolene drops relieved the condition very much. In the end with all these cases it came to a question of relieving pain. One endeavoured to maintain what sight there was, but the sight generally came down to counting fingers. He only hoped that some day—this was more a matter for the chemist than for the ophthalmic surgeon—someone would find an antidote for mustard gas. The gas shell exploded with a slight sound and might easily explode before the men concerned were aware of it. In the last war, of course, the man who got it worst of all was the gas guard in front of the trenches, who gave the alarm

when he smelt the gas, but he could not wear his gas mask before this, otherwise he would not be able to smell it.

As to recognition of these gases :—

*Phosgene* gives a choking feeling with a smell of musty hay.

*Chlorine*, irritation of eyes and throat and smell of bleaching powder.

*Mustard gas*—dichlor-ethyl-sulphide—called also “Yperite,” burning feeling of eyes, much watering, and pronounced smell of garlic.

*Lewisite*, no effect upon eyes but very deadly with a distinct smell of geraniums—“Geraniums look pleasant in a flower bed but beware their smell in war-time, if its *Lewisite* you’re dead.” Gas in “cloud” affects the greater numbers in any form of fighting, but in “shell” in trenches or buildings is a more insidious and deadly danger, with worse after-effects.

#### *The Delayed Action of Mustard Gas and the Treatment*

**Mr. T. J. Phillips** said that before discussing the clinical signs and treatment of mustard gas keratitis, he wished to draw attention to some interesting points in histories given by patients. There was a marked similarity in histories of the men concerned. They all said that following the gassing they were unable to open their eyes for about a week, and that they were having treatment as in-patients in hospital for four to six months. When at last discharged from hospital, they were free from trouble for about ten to fourteen years. The onset of the delayed keratitis was marked by either one or all of the following three symptoms: lacrimation, photophobia, and failing vision. These points in the history were given to him by all the patients except two, who stated that they had had treatment for a short period, one in 1924, the other in 1926. Both these cases were cured by lotion and ointment, no mydriatics being used; but probably they were cases of conjunctivitis. All cases gave a history of having had hospital treatment for a very long time, averaging about four months, and he understood from Mr. Whiting that this was unusual in the last war. One might deduce from this that if a man was gassed with such severity that it took many months for his eyes to heal, the case was one that might turn out later to be a delayed keratitis such as was now being discussed.

Other points of interest arising out of the history of these men were that quite a number of them were in cellars of houses when gassed, and that the action of the gas when the men were in a building appeared to be instantaneous. This point was brought out by one history of a man who was in a cellar during a bombardment. He was wearing his mask, as he had recognized the gas shells by the characteristic noise they made when they exploded. After a shell had burst inside the cellar he tried to get out and, crawling under a fallen beam in the dark, his mask was caught up in a splinter and was pulled off, exposing half his face, and one eye. He replaced his mask at once. He said his eye was only exposed for a few seconds. Now he had a typical gas keratitis in that eye only. Heat seemed to make the gas vaporize and rise from the ground. There were no histories of men having been gassed if it was raining or if there was a ground mist. Two instances brought out this point. A man was warned by an officer that there was mustard gas about. He had felt no ill-effects although he had been there for some hours. A fire was then started in a brazier, and within an hour of lighting the fire his eyes began smarting and streaming with water. The second case was of a man who had to go forward to some advance trenches and in doing so had to cross a narrow ravine. He stayed in the trenches for about two days, during which time it rained heavily. When returning across this narrow dip in the ground the sun was shining and he saw a mist rising. He thought this was gas, and put on his mask, but his eyes and chest were already

affected. The patient felt sure that no gas shells had fallen into the ravine, and no gas had come over the trenches during the two days he had been there.

He was quite unable to elucidate from any of these men that the gas was in droplet form. Some said that nothing was seen or known about it until it was smelt, and then it was too late. Others said it looked like low-lying smoke. From these histories it seemed evident that if one was in a gas bombardment it was safer to be in the open than in a building, especially an underground cellar; that the mask should be worn as soon as the bombardment started as the action of the gas was practically instantaneous, and that there was less danger when the ground was wet and when there was no sun.

*Effect and value of previous treatment of delayed keratitis.*—Most of the men seen by him had been having treatment elsewhere for months if not years. The commonest form of treatment that they had had was usually saline irrigations, instillation of mydriatics, and lubricants. Others had had repeated silver nitrate painting of the lids and cocaine ointment. A few had had repeated or single tarsorrhaphy.

The cases that did best of all were those that had had tarsorrhaphy up to the time of fitting a contact-glass. The visual results in these cases were usually good. Those treated by mydriatics and lubrication and pad and bandage also gave good visual results. Those treated by lubricants alone did not do so well as they appeared to have more deep infiltration than those treated by mydriatics. Cases giving most trouble in the healing of their corneal condition were those treated with strong antiseptics such as silver nitrate and oxycyanide lotion and cocaine ointment.

*Clinical signs of the acute stage.*—In the acute stage, that was when there was active ulceration of the cornea, the lid edges were moist and red, and along the lid margins and at the inner canthus was seen a white secretion. This was not a conjunctival discharge due to infection as was thought by some who treated the lids with silver nitrate and irrigated with oxycyanide, but a collection of desquamated epithelium.

There was absence of severe ciliary injection such as one would expect in an eye having a corneal lesion of this severity, but great congestion of the larger conjunctival vessels. There were also one or two areas of ulceration staining readily with fluorescein. These ulcerated areas were always in the lower half of the cornea and usually at about "5 and/or 7 o'clock". The ulceration usually started in an infiltrated area or in a place that had healed from previous ulceration.

If the cornea was examined by the slit lamp, the ulcerated area was seen to have sloping sides and swollen epithelium around the periphery of the ulcer. In all layers of the cornea dark lines were seen. These appeared like cylindrical vacuoles and might be described as tubes. Some of them appeared to be branched. They lay in all directions crossing one another at any angle. They were more numerous and more easily seen in the central part of the cornea than in the periphery. He thought they might be swollen nerve fibres, but he had no proof.

In this stage folds in Descemet's membrane were numerous and easily seen. When these folds appeared, the tube-like structure just referred to could usually be seen, but he had noticed the branching tubes in cases that had no folds in Descemet's membrane.

There were usually signs of iritis such as posterior synechiæ and dilated iris vessels.

*Clinical signs in the chronic stage.*—One of the most important physical signs seen in the chronic stage was the condition known as "marbling". There was an absence of small vessels, leaving large areas of the sclera bare, and here and there was seen a large vessel distended and becoming more tortuous as it neared the limbus, ending in a small corkscrew. The cornea was insensitive to cotton-wool touch, even in the upper third. Superficial corneal damage was always in the lower third and usually worse at "5 and 7 o'clock" than at "6 o'clock". He had not seen superficial

ulceration of the upper third. Deep corneal damage shown by infiltration was usually below and spread upwards. He had seen the whole cornea infiltrated. The slit-lamp in some of these early chronic cases showed grey branching lines with fluffy edges, looking like the marks made by a skate on fresh ice. These were seen in all the corneal layers and might be the end-result of the so-called "tubes" already described; they never disappeared. There were usually signs of old iritis such as posterior synechiæ and pigment on anterior lens capsule. A point of interest was the absence of lens changes.

#### *Treatment*

The best treatment for the acute eyes was irrigations with saline or potassium iodide 2%; instillation of hyoscine,  $\frac{1}{2}\%$ , the lids should be strapped, and pad and bandage applied. He had found hyoscine heal up ulcers that had refused to improve on atropine. He thought it better to apply hyoscine and paroline, rather than use oily hyoscine or atropine. He believed the castor oil used in making up oily atropine and hyoscine to have some harmful effect on the healing epithelium in these cases. He had tried cod-liver oil and halibut oil and did not think it any better than paroline.

If the ulceration persisted he gave the patient the above treatment and also two doses daily of fifteen minutes each of short-wave diathermy. He found this would usually heal any ulcer in seven to ten days, and he was now talking of ulcers that had been active for weeks if not months. He found that the best results were obtained if a wave-length of about 16 metres was used. He had tried the ultra-short-wave machines of 6 metres, but found them not so good as those of a little higher wave-length. The best results had been obtained on the "Thera-Coupler", a machine that was admirably suited for eye work. It was easy to handle, gave very good clinical results, and would stand up to hard work. The machines that he used were being run continuously for about eight hours every day. The action of this diathermy was twofold: it helped to clear up any iritis and thus gave the cornea a better chance of healing, and it produced a marked vasodilatation of the eye. In his opinion the corneal condition in these delayed cases was not produced by the direct action of mustard gas from ten to fifteen years ago, but was the outcome of the gradual cutting off of the blood supply due to the action of the gas on the vessels themselves and on the subconjunctival layers. He thought that some of the vessels were destroyed and the smaller ones partially or completely obliterated by subconjunctival fibrosis. This subconjunctival fibrosis was first seen by Dr. Dallos. When fitting men with contact glasses one very soon found that a glass might fit the eye while the patient looked in one direction, and yet if he moved his eye the glass would fit very badly and sometimes be lifted off the conjunctival surface. He took this problem to Dr. Dallos who showed him that it was produced by fibrous bands under the conjunctiva which became raised when the patient moved his eye in a certain direction. This fibrous reaction was always more marked on the nasal side and in the lower part, but was sometimes seen to a less degree above.

Two other findings seemed to confirm the idea that the corneal condition was due to a lack of blood supply. During the fitting of contact glasses the conjunctiva became red and swollen, due to manipulating the glass in the eye and this had cured a corneal lesion in the absence of iritis with no other treatment. He had massaged the lower half of the globe with an iris repositor for three to four minutes daily until a congestion was produced, and this also had brought about healing of the corneal lesion.

When these methods had failed to cure the corneal lesion, but had quietened the eye, he then gave the lesion three or four doses of local ultra-violet light. This was best done with the projecting lamp designed by Sir Stewart Duke-Elder. It had a stimulating action on the epithelium and would often induce it to grow over a naked area.

*Contact glasses.* (1) *Optical uses.*—As was expected, an eye with impaired vision due to surface irregularity was greatly benefited by contact glasses. The sort of improvement that one obtained was something of this order. Those with less than  $\frac{6}{50}$  improved to  $\frac{6}{24}$  —  $\frac{6}{6}$ . Those with  $\frac{6}{36}$  and  $\frac{6}{24}$  improved to  $\frac{6}{12}$  —  $\frac{6}{6}$ . Those with  $\frac{6}{18}$  and  $\frac{6}{12}$  improved to  $\frac{6}{12}$  —  $\frac{6}{6}$ .

These improvements in vision had been maintained by those who wore their glasses regularly and came up for periodical inspection. Some of these had attended for more than two years. There had been some regrettable cases where the vision had markedly deteriorated owing to delay by the Ministry of Pensions in the sanction of fitting, and also in one case which refused treatment and contact lenses. Great difficulty had been experienced in making some hospital patients wear their lenses as they were afraid of losing their pension. All the private cases that he had were wearing their glasses with satisfaction and gratitude.

(2) *Therapeutic uses.*—Whereas visual benefit by contact lenses might be expected, one would rather doubt the therapeutic action of such lenses on an already pathological cornea. The first case ever treated by contact glasses was a case belonging to Mr. Goulden. This man had advanced corneal lesions and was under the speaker's care, having exposures of local ultra-violet light. He had been impressed by two cases treated by tarsorrhaphy and thought it would be worth trying to protect this eye, not by tarsorrhaphy, but with a contact lens. In those days there were no such refinements as the fitted Dallos glass, and so an ordinary circular Zeiss glass was put into the eye for about two hours at a time with an interval of two hours between each occasion. The man developed an acute bronchitis and was moved to another hospital without his knowledge. Two days later he was sent for by the resident medical officer of that hospital to see "a man from Moorfields with a glass in his eye". The eye was very congested; with difficulty he managed to remove the lens and on staining the cornea found the ulcer to be healed. It might be of interest to add that some months later Mr. Goulden removed a calcified plaque from the other cornea. A contact lens was put in the eye after operation. The eye was healed, the man leaving the hospital in a few days. It had never broken down and his vision was improved from hand movements to  $\frac{6}{24}$ .

Of seven private patients only two had had small recurrences, both of which healed in a few days with diathermy, hyoscine, and the wearing of their lenses filled with paroline and not saline.

It was rather early to tabulate all the hospital cases, but of seven fitted in 1937 none had had recurrences. He hoped later to publish all the cases treated at Moorfields.

In conclusion he urged that these cases be fitted with contact lenses as early as possible even if there was no evidence of activity, as this was a sure way of protecting an insensitive and ill-nourished cornea. He wished to thank Miss Watson, the lady almoner at Moorfields, who worked without sparing herself to persuade the Ministry of Pensions to allow the fitting of these men; also Dr. Dallos for his valuable help in tackling this difficult problem. Without his skilful technique and experience as an ophthalmic surgeon one would not have been able to have fitted these men with such success. He was indebted further to all the Moorfields surgeons who had entrusted the care of their patients to him and allowed him to publish this account of their cases.

**Mr. Humphrey Neame** said that much difficulty was experienced in attempting to obtain visual improvement with spectacle lenses owing to gross irregularity of the corneal surface. Marked improvement in vision with the use of contact lenses took place in most cases, so that useful work could be done by the patient. In those

cases in which vision with the lenses was not sufficient for ordinary work, at least the patient was enabled to go about by himself and to gain more enjoyment out of life.

Some complained of blurring of vision after wearing the glasses a few hours. In some cases taking out and replacing the lens was sufficient to overcome this, in others a space of an hour or so was required before vision was restored. Comfort was attained with lenses in most cases, but in some, relief was obtained by leaving them off at intervals for an hour or so.

As to prognosis with a view to increasing opacity, presumably from hyaline deposits, and ulceration, these matters could only be decided in a few years' time. It was an astounding thing that imprisonment of the cornea in a bath of stagnant saline did good in a majority of cases. Tarsorrhaphy did not by any means always save the cornea from recurring ulceration at a time when it was practised for this complication.

It was very difficult to build a reasonable theory as to the *modus operandi* of the contact lens. He believed that Saemisch section of the cornea in an ordinary hypopyon ulcer stimulated corneal vascularization and so encouraged healing of the ulcer. One case of hypopyon ulcer that developed as a complication of mustard gas burn was treated by section without the least benefit, and the eye had to be excised. The operation did not seem to have any appreciable vascularizing effect in this case, which was shown at the Section in 1928.

In some early or slight cases a more or less triangular area of opacity was seen at each margin, somewhat reminiscent of an incipient transverse band-shaped opacity. In these the deposit was superficial. This suggested that curettage might be justifiable where there was an uneven opacity having some resemblance to a hyaline deposit, and when the vision was very seriously affected thereby so that there was not much to be lost.

**Squadron-Leader T. Keith Lyle** said that Wing-Commander Livingston, who was unable to be present that afternoon, had asked him to say a few words about some recent experimental work which had been performed in the R.A.F. This had been carried out by Wing-Commander Livingston and Flight-Lieutenant Walker. A full article on the subject would be published in the next number of the *British Journal of Ophthalmology*. In each of the cases a rabbit's eye had been exposed to one drop of mustard oil solution, and the eye had been kept open for one minute. Twenty-three rabbits had been used altogether, and had been divided into three groups. The first was an observation group of nine rabbits in which no treatment was given until the end of the first week, and then the eyes were irrigated with merthiolate of mercury in 1 : 10,000. The second group received local treatment from the commencement by irrigation with sodium bicarbonate and instillation of cod-liver oil drops. The third group was treated by intravenous ascorbic acid. In this third group six injections of 500 mgm. of ascorbic acid were given to four rabbits, and in each case the right eye was irrigated daily, while the left eye had no local treatment whatever. The final results were very similar whether or no local treatment was given in addition to ascorbic acid. The ascorbic acid was injected twenty minutes before the eye was treated with mustard oil, but latterly other rabbits had been treated two hours after the mustard oil had been put into the eye, and the results were equally good. The dosage of ascorbic acid had further been reduced from 500 mgm. daily to 100 mgm., and instead of ascorbic acid, sodium thiosulphate, a cheaper preparation, had been used, which appeared to give almost as good results. He showed a series of photographs and photomicrographs of the eyes of the experimental rabbits illustrating the extensive corneal damage done by the mustard oil, the desquamation of the surface epithelium, infiltration of the substantia propria, loss of light reflex, and the development of an ulcerated surface

on the cornea, also photographs showing the great improvement when ascorbic acid had been used. In the series of rabbits treated with ascorbic acid the reaction in the eye was minimal, there being practically no discharge or inflammation of the eyelids, and only a slight residual corneal opacity.

**Flight-Lieutenant Walker**, who was associated with Wing-Commander Livingston in this research, described the reasons which led them to approach the problem from a new angle by attempting to create or reinforce a "tissue barrier".

Wing-Commander Livingston had described two stages in mustard gas injury—the first stage of immediate focal involvement of the epithelium and the second stage of spread, in which there is a deep spreading keratitis. The first stage occurs immediately upon the application of mustard and there is no practical way of preventing it. The second stage cannot be affected by superficial applications to the cornea.

Liquid mustard does not remain stable in the secretions of the eye. This was shown by the exposure of an eye to mustard and after fifteen minutes removing the secretions of the eye and instilling them into another eye. The second eye was quite unaffected. Therefore it was concluded that chemical antagonists to mustard, applied to the eye, could have little value.

There has been much speculative biochemistry on the subject of mustard gas injury, but as yet there is little understanding of the subject. If mustard does not remain in the tissues as such, the delayed spreading lesion of the cornea that recurs over many years must be ascribed to some biochemical interaction with the corneal tissue. A feasible, but hypothetical explanation, is that the free amino group of the protein reacts with the liquid mustard, producing a heterocyclic ring, which is stable, toxic, or possibly an enzyme poison.

Ascorbic acid is a substance that is present in the lens-cornea system in fairly high amounts, and it has the unique property of being able to act as a hydrogen donator or an oxygen acceptor. It seemed worth while investigating the effect of ascorbic acid, given intravenously, in interfering with the reaction between mustard and the corneal tissues and thus preventing the stage of spread. The results of this have been described by Squadron-Leader Lyle, and since the initial work they have been confirmed and extended.

It is possible that other substances which are cheaper and more easily available than ascorbic acid will have the same effect. Sodium thiosulphate has been used, but the observations made have as yet been insufficient for any conclusion to be drawn. Wing-Commander Livingston saw the early cases of the sodium thiosulphate group and stated that while the substance is of very definite value, the results fell short of ascorbic acid treatment, but further work on the subject is necessary.

A communication was read from **Professor John Eyre, M.D.**, Consulting Bacteriologist to Guy's Hospital, saying he had just been testing with Professor Gibson, the mustard gas expert, a commercial preparation labelled Pilgrim Tablets, and they had satisfied themselves that as a decontaminant for skin lesions it was superior to either of the official preparations, viz. Antigas Ointment No. 2 and Chloramine-T Ointment, neither of which must be allowed to come into contact with the eye. [Further information may be obtained either from Professor Eyre or from Editorial Office, *Proceedings*.]

**Mr. E. Wolff** referred to the usefulness of the preparation chloramine-T ointment, made by the firm of Cusi, which was not very irritating to the eye. He did not know whether that could be of use against mustard gas.

**Sir Richard Cruise** asked what were the statistics of blindness resulting from mustard gas.

**Mr. C. B. Goulden** replied that 51 had been blinded and 180 were receiving pensions on account of their eyes.

**Dr. C. I. B. Voge, Ph.D.**, said that he had been working for some two years on the special powder referred to by Professor Eyre and Professor Gibson. This preparation had a neutralizing effect on mustard gas and could be used on and tolerated by the eyes.

**Mr. Lindsay Rea** said that figures recently published showed that of 700 war pensioners in Sheffield only one had been blinded through gas. Tear gas as a rule resulted in no permanent eye trouble. With regard to what had been said in that discussion about the treatment of mustard gas cases, he felt that it had been very unhelpful to the battalion medical officer or the medical officer at the first-aid post, the field ambulance, or the casualty clearing station, through which the gassed man had to pass. The Germans suffered greatly from mustard gas in the last war and in a translated edition of their Rules obtained in 1918 they advocated the use of an alkaline 10% lanoline ointment to be applied immediately. To-day the same German official book recommends cod-liver oil made alkaline with some glucose added. One method recommended by Bonnefon was the use of 800 parts saturated magnesium sulphate with 200 parts syrup of sugar, but this treatment was that which had been used for many years in India as well as in the Lock hospitals of London for acute blennorrhoea of adults. The osmotic action produced was marvellous: the fluid became turbid in a few minutes, especially if used in Batten's hydrophthalmoscope.

But all treatment could do no more than ameliorate if the man's condition had been neglected for the first twenty-four hours, and at present there was nothing provided for immediate application, a treatment which the man himself could apply when perhaps he was lying in a gas-saturated shell-hole for hours. Mr. Lindsay Rea's belief was that a small tube of alkaline 10% ointment and a small tube of ophthalmic boracic ointment could easily be carried and the man himself could apply these until more skilful treatment was available. There was no chemical action between sodium bicarbonate and mustard gas, and it was probable that an oxidizing agent which destroyed the effect of the gas would do more harm by the liberation of free hydrochloric acid. The eye was not a test tube and therefore must be treated purely from an ophthalmic point of view.

**Mr. M. H. Whiting**, in replying to the discussion, said that the theory that there was a pool of tears in which the gas dissolved could not be upheld. The lacrimation was so extreme and had continued over so long a time, that the solution would have been so dilute that it would have taken an expert chemist to recognize that there was anything there at all.

The cases with the dreadful results which had been related to the Section were a very small number out of the 80,000 to 100,000 cases of men who were gassed during the war; and when it was remembered that some of these serious results might have been caused by a mistake in treatment or neglect of treatment—or at least that this might have been a contributory factor—he thought the matter was seen in better proportion.

The fibrosis and dilatation of vessels that Mr. Phillips had mentioned were features that he had himself seen in cases quite shortly after the gassing had taken place.

The experimental investigations which Squadron Leader Lyle had related rather resembled those which had been carried out by an American chemist, he thought at Major Derby's instigation.

**Mr. R. E. Bickerton**, who also replied, said that many of the cases gassed in the last war by lacrimatory gas were men who had occupied gun positions and the enemy had tried to gas them out, but most of the other cases were those of men who were gassed in the testing chamber for anti-gas masks and instruction. Among these no very serious effect was seen.

Castor oil, if fresh, had a much more soothing effect than paroleine, but cod-liver oil was better than any of the others, where it did not smart too much. The drawback was that one could not dissolve cocaine or any of these alkaloids in cod-liver oil.

## Section of Otology

President—T. RITCHIE RODGER, M.D.

[February 2, 1940]

### DISCUSSION ON AUDIOMETRIC TESTS AND THE CAPACITY TO HEAR SPEECH

**Dr. A. W. G. Ewing**<sup>1</sup>: During recent years audiometers have been increasingly used for the detection of defects of hearing and for measurements of deafness. In the report published in 1938 of the committee appointed by the Board of Education and the Ministry of Health, to study problems relating to children with defective hearing, the use of audiometers was recommended for the ascertainment and measurement of deafness. Before the publication of that report audiometers were being regularly employed by the school medical service in London and in a number of other cities, towns, and county areas. In America laws have been enacted in the States of New York and Pennsylvania making audiometric tests of school children compulsory. Audiometers have been used in making a very extensive survey of the incidence of defective hearing amongst school children in New York City (Caplin, 1937). An early type of audiometer was used in the survey of American schools for the deaf, under the auspices of the National Research Council in 1924–25 (Day, Fusfeld, Pintner, 1928). Audiometric tests have proved their worth in other specialized fields. A recently published paper describes the use of a pure-tone audiometer to detect the onset of deafness amongst aviators (Dickson, Ewing, Littler, 1939). It appears that they are liable to a form of occupational deafness which results in a typical curve for loss of hearing with greatest loss at or about the pitch of 4096 ~ (C<sup>5</sup>). It is significant that this deafness is very readily recognized from the results of tests by pure-tone audiometer but not in its early stages by a whisper test.

A pure-tone audiometer was first put into regular use in our own department eleven years ago, not to detect deafness, but to find whether children known at that time as deaf and dumb, possessed any potential capacity to hear. Educational experiment had shown us that even when a deaf child could only be enabled to hear speech to a relatively small extent, the exploitation of that capacity in association with lip-reading was a better way of teaching him than lip-reading by itself could ever be. During the same period the employment of the thermionic valve in wireless receiving sets was soon followed by its use in hearing aid apparatus. Thus for the first time it was possible to amplify speech to high levels of loudness with a very good standard of accuracy. We carried out experiments to make sure that the use of powerful hearing aids was safe for patients with aural lesions, and to find at what levels of loudness they could hear best (Ewing, Ewing, Littler, 1936). It was necessary to study all the factors involved in the essentially human capacity of hearing and understanding speech.

<sup>1</sup> From the Department of Education of the Deaf, University of Manchester.

It is well known that the basic features of our telephone systems are the result of Alexander Graham Bell's efforts to make a hearing aid for his wife, who was deaf. The science of telephone engineering has more than repaid that debt to the deaf; valuable pioneer work has been done by other physicists and by psychologists, but audiometric methods of testing hearing and the greater part of our knowledge of the physical nature of speech sounds are due to research undertaken for the telephone industry.

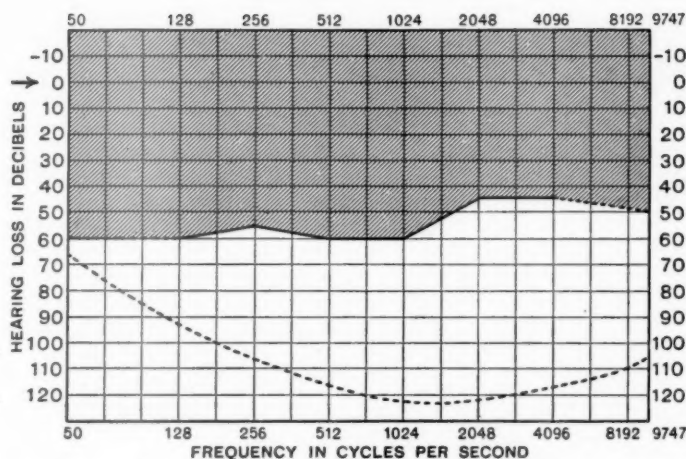
An efficient pure-tone audiometer is an instrument of precision. Fresh evidence of this was given in a paper published by Dr. A. A. Hayden of Chicago in the *Journal of the American Medical Association* of August 13, 1938. Tests of 22 patients were made with three audiometers, each of a different type and made by a different firm. The results, expressed in composite hearing curves, agreed closely. The extreme range of variation between the points on the curves at any one frequency below 4096 ~ was 15 decibels. Above that frequency the threshold could not be reached in every case. The observed variation in the results obtained was small. In part it may have been due to difference in the types of telephone used, rather than to difference in the calibration and output of the audiometers themselves. Some experiments by our lecturer in acoustics, Dr. T. S. Littler, have shown that when a telephone is applied to the ear resonance within the external auditory meatus must be taken into account (Littler, 1939). There are two sources of variation in the resonating system thus formed. There are individual variations in the size and shape of the external auditory meatus and the volume of air enclosed depends also on the type of telephone used. The extent to which these factors influence the results of audiometric tests may be small but they call for further investigation.

Joint committees of otologists and physicists have been set up by the responsible authorities here and in the United States to formulate standard specifications for audiometers. Pending the issue of their reports it would not be right to pass over this question of the standardization of audiometers without saying that the work which they have undertaken was fully necessary. Good and reliable audiometers have been produced by firms with an able research staff. All the instruments offered for sale, however, are not by any means of equal merit. There is also the question of breakdowns. So long as the risk of these exists warning should be given by the makers about the most likely defects, which in the absence of technical information may pass unnoticed.

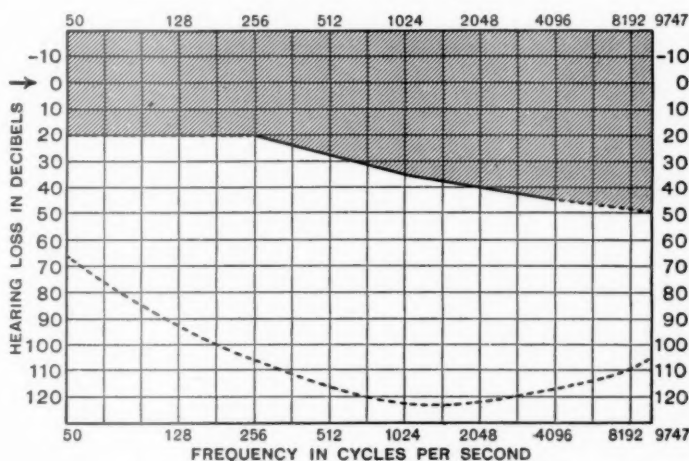
The classification of audiograms has naturally received much attention from investigators and in the main their conclusions agree. In our own Department we have made tests to compare the capacity of over 1,000 deaf patients to hear speech with measurements of their acuity for pure tones. On this basis two main types of audiogram are shown to be distinctive.

The first is that in which loss of acuity for sound heard by air conduction is either (1) approximately uniform throughout the range of pitch involved in speech, or (2) is greatest in the middle of that range. In either of these cases the essential feature as regards capacity to hear speech is that effective hearing for upper tones is retained. Hearing by bone conduction in such instances deviates less from normal, when measured by the decibel scale, than hearing by air. This type of audiogram has been recognized by medical investigators such as Dr. P. M. T. Kerridge (1937) in this country, and Dr. J. H. Jones working with Professor V. O. Knudsen (1938) in America, as indicative of deafness due predominantly to middle-ear trouble, resulting in conductive loss.

The following hearing curves are those for the better ear, by air and by bone conduction, of one of the patients of this kind referred to our clinic from hospitals and by private doctors for advice about hearing aids and lip-reading. Results of hearing for speech tests are included with the patient's history. The methods used to make them will be discussed later.



Audiogram 1. Patient No. 1.—Air conduction test.



Audiogram 2. Patient No. 1.—Bone conduction test.

Score in speech test	{	unaided hearing	..	..	Vowels	Consonants
					$\frac{10}{10}$	$\frac{10}{10}$
		with valve aid	..	..	$\frac{10}{10}$	$\frac{10}{10}$

Age : 45 years.

History : Deaf for eight years. Diagnosis—dry catarrh.

Tinnitus : Yes.

Own speech : Too quiet to be easily heard in average town noise background. No defects in pronunciation.

Notes : Paracutic. Reported could hear broadcast speech when very loud.

In these and succeeding audiograms the zero base line for both air and bone conduction charts is the same, and represents the average threshold of minimum

audibility for a group of normal listeners, measured in a sound-proof room. The semicircular dotted line towards the bottom of the chart indicates the maximum level of loudness at which sound heard by air conduction is tolerable to persons of normal hearing. Sound of greater intensity may cause severe aural fatigue, associated with tinnitus and in certain conditions acute pain. This dotted line, sometimes called the "threshold of feeling", should not appear on the bone conduction charts. In our experiments we have not reached the threshold of feeling by bone conduction with any available apparatus, although at low levels of pitch we have felt violent jarring of the skull with a tendency to vertigo.

With normal subjects listening in a sound-proof room a greater expenditure of energy is required to stimulate the sense of hearing by bone than by air. Dr. Littler and I have found that we could hear speech and music satisfactorily with bone conduction receivers connected to a high quality microphone and valve amplifier but that even in a sound-proof room the loudness level was appreciably raised by plugging the external meatus of both ears.

The second distinctive type of audiogram is that in which both air and bone conduction curves slope more or less steeply downwards from left to right of the chart because hearing loss is progressively greater the higher the pitch of the test sound used. Statistics about high-tone deafness have been published from Johns Hopkins Hospital, Baltimore (Crowe, Guild, Polvogt, 1934). A loss of acuity exceeding 60 decibels at 4096 ~ was almost invariably followed by a pathological finding of damage in the inner ear in the basal turn of the cochlea. The following are curves, for air and bone conduction respectively, of the better ear of a patient of 15 years of age. The curves for the other ear are similar but rather worse.

*Patient No. 2.*—(a) Hearing by air conduction. (Audiogram 3, p. 25.)

(b) Hearing by bone conduction. (Audiogram 4, p. 25.)

Scores in speech test, after six years' tuition :—

	Without aid or lip-reading	With valve aid, without lip-reading	With lip-reading without aid	With valve aid and lip-reading
Vowels ..	24	76	81	94
Consonants ..	7	46	66	86

History : Pneumonia at age of 6 weeks. No deafness in family.

Otologist's report : Drums normal in appearance. Rinne's test positive. Very marked loss of bone conduction.

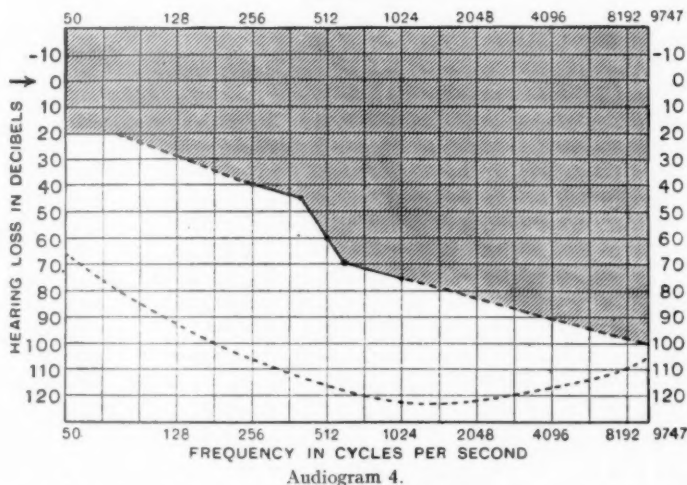
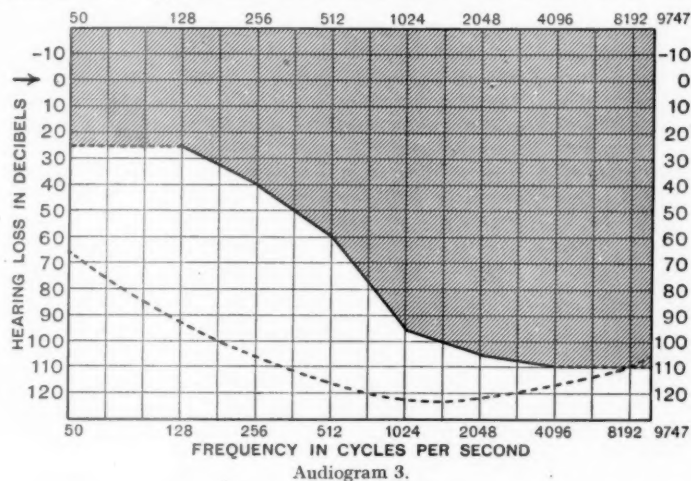
Own speech : Very little and imperfect speech when first seen at 9½ years of age. Individual tuition by means of lip-reading with hearing aid was then begun. Speech was approximately normal when tuition ceased at 17 years old.

It is noteworthy that patients with this type of audiogram often retain fairly good, sometimes even approximately normal, hearing for lower tones. Despite that there is always a limit to the accuracy with which they can be enabled to hear speech, even with the kind of aid that suits them best.

Besides these two distinctive types of audiograms there are many characteristic of what is sometimes described as mixed deafness. The air and bone conduction readings for lower and middle frequencies are approximately of the even loss kind, but deafness to upper tones is much more severe. Such audiograms are often obtained from tests of patients with an otological diagnosis of middle-ear disease and a history of long-standing deafness.

There is a technique which we have found useful for determining the extent of any patient's capacity to hear speech. It combines (a) information taken from his audiogram with (b) the results of standardized tests of his ability to hear vowels and consonants in syllables.

This procedure was originally founded on the results of investigations in connexion with telephone engineering. By using sound filters in a high-quality telephone system, or in an electrical system for reproducing gramophone records, it is possible to limit the range of pitch over which sound is heard by the listener. It is readily demonstrable that suppression or much reduction of the loudness of sound in the



upper half of the speech range interferes very seriously with the distinctness with which speech is heard. A voice is audible but the words are difficult to recognize. The experience is somewhat like that of listening to someone talking in a distant room with the intervening door closed. Consonants suffer more than vowels, but vowels lose much of their characteristic quality.

Both are sounds of a complex nature, but vowels lend themselves more readily to

physical analysis. The type of diagram known as an acoustic spectrum is a particularly clear method of showing the results of this.

Thus represented the components of the vowel EE, as in "eat", are seen to extend over a range of from four to five octaves. The strongest components are produced below the frequency 600~ and round about that of 2400~. This pattern is characteristic of the vowel EE, irrespective of its utterance by a male or a female voice, of the fundamental tone on which the vowel is said or sung, and of features peculiar to the utterance of the individual speaker. Similarly the vowel AH as in "park" is distinguished primarily by a group of strong components at or about the frequency 1000~.

Suppression of their high-pitched components, say all those above 1000~, destroys the most distinctive characteristics of certain vowels. The effect of similar suppression on consonants is even more drastic since, in the main, consonants are weak sounds of high pitch. Here then we have an explanation of the serious extent to which high-tone deafness reduces the capacity to hear speech.

By contrast, filtering out the low-pitched components in speech, below 1000~, leaves it still intelligible, although weak in intensity and chirrupy or reedy in quality.

It has been shown in Bell Telephone Laboratories that suppression of sound in speech above 1000~ leaves it only 40% intelligible to listeners with normal hearing. The converse experiment, suppression of components occurring below 1000~, affects its intelligibility hardly at all, it is still heard with an accuracy of 95% (Fletcher, 1929).

Tables showing the average loudness levels of speech, music, and noise, in typical conditions, have been published from the National Physical Laboratories by Dr. G. W. Kaye (1937) and from other sources. The reference point of these tables is the loudness of a tone of 1000~ as heard by normal listeners. They hold good for patients suffering from even or relatively even amounts of deafness throughout the speech range. Our first step on examining the audiogram of such a patient is therefore to note his hearing loss by air conduction in the better ear at 1000~ or 1024~. At this level of pitch the average loudness of conversation as heard at a distance of one metre from the speaker varies from 40 to 70 decibels above the normal threshold of audibility. The extent of this variation is chiefly due to the great difference in loudness between the louder vowels and the weaker consonants. A patient with an audiogram of the even loss type and deafness amounting to 50 or 55 decibels at 1024~ will probably fail to hear many of the weak components when listening with the unaided ear to conversation in a quiet room. In a motor car or in a train, speakers with normal hearing raise their voices the better to hear themselves speak. Usually they also talk more slowly and distinctly, thus in two ways the patient with even loss is helped to hear in a noisy environment.

The fundamental distinction between the effects of inability to hear lower and higher tones respectively holds good in conditions of deafness. A patient with a diagnosis of predominantly middle-ear defect and relatively uniform loss can hear speech with considerable accuracy provided that its loudness level is at least 30 decibels above his threshold of audibility. This accounts for the striking degree of benefit which such patients often obtain from the use of modern valve hearing-aids giving good reproduction of sound in the upper half of the speech range. The results of hearing for speech tests with Patient No. 1, given above, are a good illustration.

In high-tone loss, on the other hand, the accuracy with which the patient hears speech is reduced out of all proportion to the diminution in its apparent loudness. He may hear a foghorn a mile distant but fail to follow average quiet conversation uttered 36 in. from his ear.

How drastic is the loss of capacity to hear speech which may result from high-tone deafness is shown by the vowel and consonant scores of Patient No. 2. These also show, however, that a most valuable measure of alleviation was possible through the use of a suitable valve aid in combination with proficiency in lip-reading. It is

noticeable that the combination of lip-reading with the aid gave far better results than either means of help by itself. There is an increase of the consonant score from 7% without the aid of lip-reading to 86% when both means of alleviation are allowed. This increase becomes all the more significant when the usual weighting is applied to the consonant score. In connected speech consonants occur much more often than vowels, and it has been found that a formula can be applied, the simplest version of which is  $I = VC^2$ , where  $I$  is the percentage intelligibility of speech syllables, and  $V$  and  $C$  the percentage scores for vowels and consonants respectively.

In the condition described above as that of mixed deafness, loss of capacity to hear speech is a sum of the effects of the two factors, i.e. even or relatively uniform loss for lower and middle tones and a lesser or greater degree of high-tone deafness. Recent studies have been made of the elimination of auditory acuity which normally takes place with advancing age in individuals free from ear disease (Knudsen, 1937). It is unfortunate from the speech point of view that hearing in the upper half of the speech range is most affected. Individuals belonging to the age-group 50 to 59 years, for instance, show an average loss of acuity of 15 decibels at 2048 ~ and 30 decibels at 4096 ~. Such loss, if unaccompanied by impairment due to any other cause, does not as a rule appreciably diminish capacity to hear conversation but it is a probable source of difficulty in a public hall or other auditorium, where acoustic conditions make it difficult in any case to hear the weaker consonants (Montgomery, 1932). The combination, in cases of "mixed" deafness, of greater hearing loss than this for high tones with appreciable deafness to lower and middle tones added, results in conversation being imperfectly heard.

Finally, a brief reference should be made to methods of testing hearing for speech with the human voice itself. They are necessary in addition to audiometric tests for several reasons. Measurements of the patient's threshold of audibility do not always tell us essential facts about the response of his ears to sounds of normal conversational loudness. There is, for instance, the phenomenon which American workers have called "quality deafness". Sound, in such cases, although heard, appears distorted. An organist, for example, who has become deaf reports that below middle C the sound of his instrument still appears normal. Above it the organ seems out of tune. To him tenor voices are still satisfactory but soprano singers appear out of tune.

To test a patient's capacity to hear speech is to test much more than his capacity to hear. Ability to follow what is said depends also on familiarity with the thought and vocabulary of the speaker. It involves the activity of the whole mind and is only possible as the result of past experience. We are all, of course, familiar enough with these facts through analysis of the difficulties which arise in telephone conversations. In listening on the telephone intelligence is brought into play, to relate imperfectly heard phrases or proper names to their context. Then, as in lip-reading, a whole sentence generally offers more clues than a single word.

For this reason clinical tests of the capacity of deaf patients to hear speech, although needful, cannot be as precise in their results as those with a pure-tone audiometer. A trained worker in controlled acoustic conditions can obtain scores from voice tests indicating approximately the extent to which vowels and consonants are heard correctly. This procedure is invaluable as part of the basis for giving advice about the choice of a hearing-aid. The score obtained with the unaided ear can be compared with the scores obtained with different aids.

The selection of material for such tests is not easy. In the telephone industry nonsense syllables have proved to result in the most searching form of test (Fletcher and Steinberg, 1929). When they are used the influence of context and of the mental factors generally is reduced to a minimum. Previous practice is needed, however, if a very high degree of accuracy is desired. In testing deaf patients some workers like Mr. D. B. Fry and Dr. Kerridge (1939) are using systematically prepared lists

of words and phrases. For this purpose we ourselves rely at present on a graduated test, beginning, with numbers, as a form of practice, followed by tables of simplified nonsense syllables and by connected speech at varying distances. But before any voice tests are made we first obtain the more precise measurements that are possible with a pure-tone audiometer. These measurements, we have found when interpreted by the principles which I have described, indicate clearly the patient's capacity to hear speech, and, as a rule, the type of aid most suitable for him.

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**Mr. W. M. Mollison:** It was hoped that the advent of the audiometer would solve all the previous difficulties and confusions of hearing tests. Previously tests for hearing had been at the caprice of the individual observer; for this reason the strictly comparable audiogram which expressed in a curve the quantitative loss of hearing was rightly hailed as an enormous step forward.

The audiometer, however, has some drawbacks; its range is limited at both ends of the scale. Its lowest tone is that of 64 d.v. or 128 d.v., and its highest 10,000. There are cases in which the loss of the lowest tones (16 or 32 d.v.) is the only sign of slight deafness, a condition sufficient to produce middle-ear vertigo, and occasionally the only sign of otosclerosis in an apparently sound ear, the disease having attacked one ear much earlier. Inability to test the upper end of the scale prevents the discovery of high-tone deafness; I refer particularly to those interesting cases of familial loss of hearing of the highest tones, with consequent inability to hear the sounds made by bats and by some birds, by various members of the family.

But the more serious objection to the audiometer lies in its lack of practical application. After all, the lack of perception and interpretation of spoken words is what interferes with social life, not the lack of hearing of pure tones. As the result of studying large numbers of audiograms it should be possible to state the amount of conversational disability in terms that the man in the street could appreciate. This is at present not the case without detailed analysis not easy for the otologist. The time is not ripe for the audiogram to be considered as a means of supplying, as it were, a diagnosis. Recently an aurist was asked to examine a man who was claiming

compensation for deafness after an accident; his report took the form solely of the audiogram he had obtained. Not unnaturally, this report was of no value to the doctor and would have carried no weight with counsel, judge, or jury.

[Some audiograms were shown to illustrate difficulties of interpretation.]

Difference in hearing of the two ears is most important, and becomes specially so in high degrees of deafness, even though the difference is small. It is disappointing that this is not always shown in audiograms.

**Mr. Terence Cawthorne:** Those who work in very noisy surroundings, or whose ears are periodically subjected to extremely loud sounds, suffer from a gradual falling off in the acuity for high tones. Formerly this state of affairs was only recognized when the high-tone loss was sufficient to interfere with the appreciation of speech. The audiometer has enabled this high-tone loss to be detected at an early stage before it has interfered with the intelligibility of speech. This may mean that if adequate measures are taken to protect the ears against loud sounds deafness may be prevented; and I firmly believe that such measures if generally adopted will cause a remarkable decrease in the incidence of so-called senile deafness.

Although Dr. Ewing has clearly shown that an audiogram is a guide to the ability to distinguish speech sounds, we cannot therefore assume that other methods of testing the auditory apparatus are unnecessary. Any hearing loss revealed by an audiogram is based on the threshold of hearing. Such a loss does not necessarily indicate a corresponding loss for the threshold for speech any more than a diminution of the light threshold for the eye indicates an inability to distinguish the printed word.

The pure-tone audiometer and the voice each play their part in the investigation of the auditory apparatus. The audiometer reveals degrees of hearing loss for pure tones so slight that they do not affect the ordinary speech test; and in the case of the severely deafened it indicates the class of hearing aid that is most likely to be of help. When we come, however, to the actual choice of a hearing aid, some form of speech test is essential.

In comparing audiograms with speech tests in a quiet room, a patient with a noticeable loss in the high tones is able to distinguish the spoken and sometimes even the whispered voice at 20 ft., although in such cases it is the whispered voice that is the first to be reduced. In the slighter degrees of high-tone loss difficulty in distinguishing the voice may only be apparent in a noisy surrounding, a state of affairs one usually tries to avoid when hearing tests are in progress.

Speech tests are helpful when the hearing loss is not severe, and especially when comparing the acuity for the spoken and the whispered voice. In the more severe degrees of deafness when the spoken voice can barely be heard the speech test does not tell us so much. A disadvantage of the speech test as a method of recording the hearing capacity is the difficulty in ensuring a uniform level of loudness for the voice. Also records of speech tests rarely indicate whether numbers, single words, or sentences have been employed to make the test. These drawbacks have largely been overcome by the use of standard words or sentences and the gramophone audiometer where series of numbers are spoken by a voice in known steps of decreasing loudness.

Finally, we must guard against comparing tests that are not strictly comparable. The pure-tone audiometer gives us valuable information about the integrity of the auditory apparatus with reference to the threshold of hearing, but this does not mean that any loss at this level is necessarily the same at the level of speech sounds.

**Mr. F. W. Watkyn-Thomas** said that whilst the audiometer was a most valuable instrument in the investigation of deafness, especially high-tone loss, he did not accept the audiometer findings as a true picture of hearing capacity. The marked divergence between clinical findings and audiometric curves had been brought out in the examples

just shown by Mr. Mollison. The audiometer was an instrument of precision, but the defective ear of the patient was not. It was otherwise in ophthalmology, where the patient either knew or did not know that a letter shown him was Z or whatever it might be. But the patient heard at one level of stimulus and at another level of stimulus he did not hear, and the investigator did not know what the patient actually did hear and could not tell how the stimulus was interpreted. Dr. Kobrak, at University College Hospital, was at the present time trying experimentally to work out this question.

He would limit himself to one of Dr. Kobrak's observations. In many cases there was a considerable difference in the threshold according to whether one reached it by starting with the sound the patient could hear and working down until the hearing disappeared, or, on the other hand, starting from zero loss and working up until the sound was heard. The width and the form of that threshold band had given facts of clinical significance.

**Mr. Scott Stevenson** said that the real truth was that a clinical picture of the deaf patient had to be arrived at, and as they worked, not in the laboratory as Dr. Ewing did, but in the consulting room and the hospital, they had to make use of all available tests. He himself always used tuning forks for the low notes, then an audiometer, and he also used speech and whisper, and if there was any question of very high notes he used the monochord. The advantage of the audiogram was that the records could be checked up a second time, and this was much more valuable than the mere statement of a personal opinion as to the reaction to the voice or whisper or the tuning fork. Clinicians had to work on the combination of their findings with these different tests, but if he himself were limited to one test only he would give up the tuning fork and the voice test in favour of the audiometer.

**Mr. C. S. Hallpike** said that if it was a question of carrying out pure-tone tests, then the audiometer was the instrument to use. Pure-tone tests were worth doing, though correlation of such tests with hearing for conversation seemed a more difficult matter. As Mr. Cawthorne had stated, audiometric tests were tests of threshold, whereas in ordinary life the ear worked at a level some 60 decibels above the threshold. Dr. Fowler, senior, of New York, had done some interesting work on that point in cases of unilateral internal ear deafness, and he found that although there might be a difference at threshold of some 30 or 40 decibels, nevertheless, if the intensity at each ear were increased by about 20 decibels, then the difference in loudness was much reduced and disappeared with further equal increases of intensity up to conversational level. In other words, although the two ears might work very differently indeed at the threshold as measured by the audiometer, at the working range of the ear the "bad" ear worked as well as the "good". This arose from the fact that the ear worked with neural units, sensory cells, and nerve-fibres, which obeyed an all-or-nothing law and was a specific instance in which threshold tests would be misleading.

In audiometric tests the signal might be considerably distorted by the ear, but so long as the patient heard at all, the correct answer would be given. Distortion would interfere much more, at any rate at first, with the recognition of the complicated wave forms of speech, which was, after all, the most important function of the ear.

**Sir James Dundas-Grant** said that though the audiometric tests represented a distinct advance they could not afford to abandon the other tests. He had considered these a great deal, and he would refer to a scheme for the measurement of loss of hearing in decibels by means of tuning forks which he had published in the *Lancet* of November 2, 1935. Anyone could see enormous value in the audiometer for testing large numbers. When it came to the individual patient there was one hearing test

which had considerable value, namely, what the friends and members of the family of the patient said with regard to the progress or otherwise of his hearing. If they said that he heard better than before, the clinician might be sure that he did so but the converse did not necessarily follow.

**Dr. H. Frey** said that while the audiometer had improved the machinery of testing it had not done away with the difficulties which were inherent in the fact that hearing tests were essays in experimental physiology and psychology. It was true that a difference resulted according as to whether the testing started from a higher intensity and worked downwards or from a lower intensity and worked upwards, but that was a difference of value which pertained to every kind of similar experiment. The threshold was not a sharp line but a broad band, and this being so, perhaps an average might be attempted.

He had found long ago with patients who had suffered from acute otitis or acute middle-ear catarrh, and who were examined regularly by speech tests, by tuning forks and to some extent by the audiometer, that long after the patient had regained his full hearing power for speech, whisper, and so on, the tuning fork tests did not become normal again. This might go on for many weeks, sometimes for months.

Audiometric results did not give a full assessment of the patient's hearing. The hearing of speech depended as much on psychological conditions as on physiological ones.

**Dr. F. W. Kobrak** said that it was very important to bear in mind the difference between testing for single tones, especially pure tones, and the combination of tones and rhythm. He had had the opportunity of testing the instrument at a hospital and had been somewhat surprised at the disparities between the findings and those obtained with the tuning fork. He had started by supposing that the tuning fork and the audiometer tests would be the same, but they were not the same, they were in fact quite different. The audiometer was undoubtedly interesting, but he could not help thinking that it would be a great mistake to accept an audiogram as a test of efficiency. There were also some valuable possibilities in the tuning fork, especially in respect to the decay of vibration, and in his view it would not be right to abandon the tuning fork in favour of the audiometer. The features of the tuning fork were so important and so closely related to the efficiency of hearing that it would be a great mistake to give the instrument up.

**Dr. A. W. G. Ewing**, in replying to the discussion, said that the remarks of speakers seemed to have proceeded on two lines; first of all, with regard to the inter-reliability of audiometric methods of testing and other methods, and how far the results of the one method were consistent with those of the others, and, secondly, to what extent the results of audiometric tests furnished information which it was necessary to have concerning the patient's state of hearing.

As to the first point, there had been a good deal of discussion regarding the relative reliability of audiometric tests as compared with tuning-fork tests. The point, he thought, was best answered in this way. If one went to the National Physical Laboratory or to the laboratories of any physical research engineer in this country to-day, one would find that reliance was placed on electrical methods of producing sound. The engineer could control his sound field by electrical methods to an extent which he could never hope to do by means of a fork.

On the question of relative reliability of the various tests, reliability was, of course, a question of whether it was possible to reproduce the results in the patient on a number of occasions. Voice tests had been very carefully investigated—most carefully of all, so far as his knowledge went, by Dr. MacFarlan of Philadelphia. Dr. MacFarlan appears to have begun by supposing that these were a reliable form

of test—he was not excluding their usefulness even if they were not reliable scientifically—and he carried out his investigations with different speakers but could not get a reproduction of the same results, so far as his memory went, within 90%. Different callers got different results, and the same caller different results on different occasions.

Dr. Ewing did use voice tests as supplementary to the audiometric tests, but it was his experience that it was only when the consonants played an important part in the voice tests that such tests were really reliable. If the importance of consonants were cut out and reliance placed on numbers which the patient could recognize, if he was intelligent, by noticing the duration of the sound, and the vowels, there was no consistency between the results obtained with these tests and those with audiometric tests. But the more searching the voice test, the more important part did the consonants play in the material used, and the nearer one got to absolute consistency between these and the audiometric tests.

A patient's capacity to hear at threshold was a test of his capacity to hear under working conditions, because, of course, in conversation the loudness had to rise to 60 or 70 decibels above the threshold for maximum distinctness to be achieved.

Mr. Hallpike had raised the question of Dr. Fowler's paper. Dr. Fowler found that when there was much greater deafness in one ear than the other a tone of a given intensity, in certain types of case, gave an experience of equal loudness no matter to which ear it was presented. When it was a question of 60 decibels above threshold, for example, the capacity of the "deaf" ear might appear to be the same as the normal ear. But he believed that was only the case in nerve deafness, not in middle-ear deafness. There was no doubt that the equal loudness curves, which had been plotted out by physicists for normal ears, held good, in internal ear deafness, for deaf ears also. The form of audiogram made by the Maico Co. gave not only intensity levels but equal loudness curves. If one had a deaf patient, supposing he could hear at all with his affected ear at 60 decibels above threshold (to repeat the previous example), the sound would be equally loud to him in his affected and in his unaffected ear at that same intensity level. But that was just a further extension of their knowledge concerning hearing. It was not in any way a handicap to the use of audiometric methods.

As regards the question of correlation, surely it might be said, as one speaker had pointed out, that every hearing test was a psychological experiment, and in clinical work it was not always possible to get exact results. But the question when different methods of testing hearing were compared was how near one could get by the different methods, and the bearing of his own work had been to show that they could get nearest to the truth by audiometric methods. There were these cases of quality deafness and there the threshold would not afford complete information of the extent of the handicap in hearing speech. Statistically he did not know how often such deafness occurred, though he hoped it would be possible to state this authoritatively within a short time, but from the tests on more than 1,000 patients—indeed, on nearly 2,000—it appeared that such cases were very few and far between. In the majority of cases if there was uniform loss one had only to look at the middle of the range and one would see at once whether the patient could be expected to hear in a public auditorium or could only hear loud speech at 3 feet distant or a whisper close to his ear. A patient with 20 decibels loss at 1,000 ~ would hear speech in an auditorium with the greatest difficulty, perhaps with so much difficulty that he would become tired. With high-tone loss there was no possibility of hearing in an auditorium really satisfactorily; the patient might endure the discomfort and strain, but he did not hear everything that was said nor anything like it.

## Section of Surgery

President—V. ZACHARY COPE, M.S.

[January 17, 1940]

### Extra-abdominal Resection of the Colon

#### PRESIDENT'S ADDRESS

By V. ZACHARY COPE, M.S.

FROM an early period in the history of abdominal surgery it has been recognized that resection of a portion of the large bowel is more difficult and dangerous than excision of a segment of the small intestine. The fixity of certain parts of the colon, the great variation in size of the bowel, the presence of surfaces uncovered by peritoneum, the possible irregularity of suture-line caused by appendices epiploicae, and the semisolid nature of the bowel-contents, all combine to make the procedure more hazardous. Added to these is the fact that most cases of colonic disease come to the surgeon on account of obstruction which has been in existence for a long time before surgical advice is sought; in such cases the bowel above the obstruction is often enormously distended by gas and the wall of the gut is frequently in an unhealthy and sometimes friable state. Though some of the earliest recorded abdominal operations were performed for the relief of such obstructions by opening the bowel above the contracted part there is only one record, prior to the introduction of antiseptic surgery, of an attempt to remove a cancerous stricture of the colon. This was the case recorded by Reybard in 1843 in which he claimed that ten years previously he had successfully excised a stricture of the colon and performed end-to-end suture of the bowel. Contemporary opinion did not regard his claims as substantiated, for, at the time of recording, neither patient nor specimen was available for examination, and similar experiments on dogs were unsuccessful. In any case the technique described was not one which could be safely adopted.

Even after the introduction of antiseptic surgery more than ten years elapsed before success in this operation was achieved. At a Congress of German surgeons held in 1878 Gussenbauer gave an account of an unsuccessful resection of the descending colon for cancer which he had performed in the previous year; he had united the severed ends of the bowel by silk sutures, but soiling of the peritoneum had occurred at the time of operation, and the patient died fifteen hours after operation. This attempt, as the record stated, "showed that the operation is not a theoretical consideration but a well-warranted surgical enterprise".

In the discussion which followed Gussenbauer's paper Schede told of an attempt he had recently made to remove a colonic growth in which he had found it impossible to bring the severed ends together and had been forced to content himself with bringing the upper end of the bowel out of the wound. His patient had died next day of "inanition". The discussion also had a contribution from Thiersch who recounted a disastrous attempt at excision of a growth of the sigmoid with end-to-end suture of the bowel; after he had united the bowel by suture a peristaltic wave occurred and gross leakage of the bowel-contents forced him to finish by performing a colostomy. Needless to say the result was fatal. In 1879 also Baum made an unsuccessful attempt at resection of a colonic growth; his effort was noteworthy in that he performed a preliminary ileostomy.

No doubt stimulated by Gussenbauer's enterprise, Martini of Hamburg the following year decided to resect a growth of the sigmoid. He summoned Gussenbauer to confirm the diagnosis and asked him to assist at the operation. Together they resected the growth but were unable to bring together the bowel-ends for purposes of suture. The lower end was therefore closed by suture and the upper end brought out of the wound as a colostomy. The patient recovered. This is the first authentic record of a successful resection of the colon (November 9, 1879). From this year onwards frequent attempts at resection were made, and it was in the following year that the surgical world was startled by the brilliant achievement of Czerny who, faced by a cancer of the transverse colon which also involved the sigmoid flexure, bravely resected both portions of bowel and, to restore continuity performed two end-to-end sutures with successful result in spite of a slight leakage for a few days.

In all these cases it was the tumour, as much as or more than obstruction, which called attention to the disease. The surgery of growths causing complete obstruction was a little slower in development. In the year following Czerny's remarkable case Thomas Bryant operated upon a patient with intent to perform a left lumbar colostomy for complete obstruction of the left colon. To his surprise he found that the growth could easily be brought out of the wound. He emptied the distended bowel, cut away the affected part gradually, and sewed the upper and lower ends of the bowel to the skin close together. He remarked, "I did this with the view of facilitating the restoration of continuity of the intestinal tract and closure of the artificial anus at some future time should the patient feel disposed to submit to the operation and the measure appear desirable, which I am disposed to question". In presenting the case he added, "I submit that the expediency of removing the strictured bowel, where practicable, is more than demonstrated". His patient recovered.

During the next ten years progress was slow though various experiments were being tried. In 1883 the first successful resection of the cæcum was performed by Maydl. Kraussold, Billroth, and Bergmann had previously unsuccessfully attempted the operation by end-to-end suture; Maydl tried another method with the comment: "Since I did not consider that it was advisable to perform immediate enterorrhaphy after resecting either a gangrenous hernia or a stricture I made an artificial anus in such a way that the ends of the intestine were partly joined together and partly joined to the abdominal wall." His patient left hospital in a month but returned later for closure of the anus; this entailed resection of 6 cm. of gut and end-to-end union. Recovery took place but a slight fistula remained.

The first attempt to reconstitute the lumen of the bowel by destroying the spur between brought-out ends of the gut was made by J. M. Barton in 1887 (America). Immediately after resecting the ileocæcal region for growth "one blade of Dupuytren's enterotome was introduced into each portion of the bowel, viz. one into the ileum and one into the colon, the two blades were brought together, and the screw run down firmly. A strong ligature was placed on the ends of the bowel, including the enterotome, to prevent the escape of faeces during the subsequent manipulation". This attempt was only partially successful for the patient was left with a slight faecal fistula, but the idea was one which was developed by others later.

In 1889 a cæcal growth was successfully dealt with by Walter Edmunds by resection and bringing out of the ends of the bowel, and in 1890 Nicolas Senn published a successful case of excision of the cæcum with anastomosis between ileum and colon by means of his decalcified bone-plates.

A great advance was made when in 1892 Oscar Bloch published his famous contribution in which he recommended the extra-abdominal resection of the colon. The suggestion had been thrown out by Heineke in 1884 that resection might be better done in three stages, and Davies-Colley had recommended leaving the bowel outside the abdomen before resecting the growth, without, as far so can be ascertained, ever

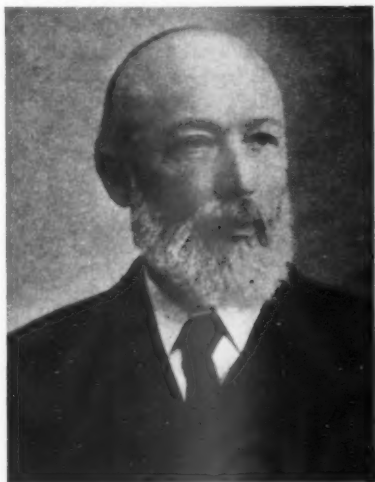
trying this method himself. It was left to Bloch to carry out the procedure. His patient, aged 36, had a growth of the sigmoid. At the first operation the affected



Oscar Bloch (1847-1926).  
(From *Ugeskrift for Læger*, 1926, 88, 606.)



Herbert William Allingham (1862-1904).  
(From *Med. Press and Circular*, 1904, Nov. 9, p. 504.)



Frank T. Paul (b. 1851).  
(From "A Medical History of Liverpool from the Earliest Days to the Year 1920," Published by John Murray.)



Joh. v. Mikulicz-Radecki (1850-1905).  
(From Fischer's "Biographisches Lexikon," 1933.)

coil was brought out of the abdomen and the bowel above the growth opened by the cautery. A month later the growth was resected and the ends of the bowel left in

MARCH—SURG. 2

the wound. Three months later the bowel-ends were freed, a circular suture performed between them, and the gut replaced in the abdomen. Recovery followed. In his paper Bloch gave a masterly summary of all previous operations performed for cancer of the colon and showed the high mortality of the operation. To him without doubt belongs the priority of recommending, upon the basis of practical experience, extra-abdominal resection of the colon in two or more stages. It is to be noted, however, that he did not cut away the exposed coil for a month after it had been brought out, and that it was three months after this that he closed the anus by resecting and suturing together the ends.

Division of the spur between the ends of the bowel was first performed in a tentative way by Walter Edmunds in a case on which he operated in 1892 and made public in 1893. This concerned a cancer of the sigmoid which was in the first instance brought out of the abdomen and prevented from receding by means of a glass tube put through the mesentery; four days later the growth was resected and the ends sutured together. The sutures gave way and an artificial anus was formed. Edmunds tried to remedy this by a lateral anastomosis between the afferent and efferent limbs of the anus, but this failed, so he then divided the intervening spur and later almost closed the faecal fistula.

Soon after this, in 1893, occurred the first instance of the modern operation of primary caecostomy followed by resection of the growth at a later date. This method we owe to Allingham (junior) who published his account in the *Transactions of the Medical Society of London* in 1894. His patient had complete obstruction of the colon. Allingham made a mid-line incision and performed caecostomy to relieve the distended bowel. Four months later he resected the descending colon and sewed the ends of the bowel together over a Mayo-Robson's bobbin. A month later he closed the caecostomy. Allingham's example was not quickly copied and for several years surgeons continued to perform resection and immediate end-to-end union with bad results. The next contribution to the subject, and one which is probably the most important contribution yet written on this subject, is that of F. T. Paul of Liverpool. Paul published his experiences in the *Liverpool Medico-Chirurgical Journal* for January 1895 and in the *British Medical Journal*, May 25, 1895. In this article are recounted seven cases of operation for carcinoma of the colon which the author truly states "represent the education of an individual surgeon". At the end of the article precise directions are laid down which have scarcely been improved upon during the intervening years. The account which Paul gave of his operations makes it quite clear that he came to his conclusions by the compulsion of experience rather than the suggestion of others. The surgeon's progress may briefly be summarized and some of his comments appended as follows. There was colonic obstruction in all cases.

May 1890: Resected growth of descending colon. Suture of ends over decalcified bone bobbin. Death from peritonitis.

February 1891: Right lumbar colotomy for obstruction with faecal vomit. Twelve days later growth of descending colon excised and the ends brought out. Patient died thirty-six hours later.

April 1892: Resection of end of ileum and caecum for growth in caecum. Suture of ileum to colon over bone tube. Death from sloughing of the colonic end. The comment is made: "The course of the case after operation indicated that he might still have survived if the ends of the bowel had been brought out, and this I fully determined to do next time."

May 1892: Growth of sigmoid excised through left inguinal incision. Glass intestinal drainage tube ligatured into each end of the bowel. Rapid convalescence. Comment by Paul: "In this case I made no attempt to restore continuity thinking that at her time of life (60) she had better be content with the advantage already gained; not that I intended to rest satisfied myself with an artificial anus in future cases."

January 1893: Growth of descending colon. Colon excised from splenic flexure to sigmoid. Glass tube put into each end of bowel. Month later ends of bowel excised and sewn together.

Death from peritonitis on the fourth day after operation. Comment: "I was naturally very much disappointed to feel that, having arrived at a safe method of removing the growth there remained a great risk to the patient's life in endeavouring to re-establish the natural channel, and I decided that it would be better to bear the evils of artificial anus if to avoid them it was necessary to take the chance of a fatal peritonitis. There remained, however, to be tried the old plan of restoring continuity by Dupuytren's enterotome to which very little risk attached. If such a method could be perfectly successful when a spur was accidentally formed by Nature, how much more ought it to be so when a spur was deliberately constructed with the object of being subsequently safely removed? I therefore thought out and determined to put in practice the following mode of operating in the next case: First excise the strictured portion of bowel as in the last two cases; then suture together the cut edges of the mesentery and the adjacent sides of the two ends of the colon, in such a manner that they would adhere together for about three inches, in the position of the two barrels of a double-barrelled gun. If this succeeded the spur might be demolished without the slightest risk of peritonitis and to such an extent as to ensure a free passage and easy closing of the artificial anus."

February 1894: Tumour in ascending colon. Exploration in middle line, then over tumour. Mesentery ligated, loop of gut brought out, five inches excised and glass drainage tubes tied in each end. Mesentery sutured and ends of bowel attached side, by side. Spur divided later by applying forceps. Later still the rosette of mucous membrane was separated from the skin, sutured together and the skin closed over it. Primary union occurred and henceforth the bowels were moved by the natural passage.

December 1894: This last case in the series was a chronic intussusception of the right half of the colon in which Paul resected the end of the ileum and the ascending colon successfully by the above method. Death occurred from uræmia a few weeks later before the anus had been closed.

Paul summarized his views by stating that in cases where the patient was in good condition, the abdomen not distended, the tumour small and the proximal end of the gut not hypertrophied, he advised immediate approximation of the bowel-ends by means of Murphy's button. "But when the opposite of these conditions prevails I strongly urge that the ends of the bowel be brought out in the manner explained and illustrated." Finally Paul described clearly the steps of the operation.

- (1) Explore first in the middle line, unless the stricture has been located.
- (2) Make a sufficiently free incision over the site of the tumour.
- (3) Having cleared away any adhesions, ligature the mesentery with the help of an aneurysm needle, and divide it sufficiently to free the bowel well beyond the growth on each side.
- (4) Let the loop of bowel containing the growth or stricture hang out of the abdomen, and sew together the mesentery and the adjacent sides of the two ends, as shown in the engraving (p. 16). See that the stump of mesentery lies beneath the bowel, where if deemed advisable, it can be drained by packing cyanide gauze down to it.
- (5) Ligature tightly a glass drainage-tube into the bowel above and below the tumour, and then cut away the affected part. (Don't cut off first, or blood will be unnecessarily lost.) Only the proximal tube is really necessary. The distal end may be closed or included in the proximal ligature.

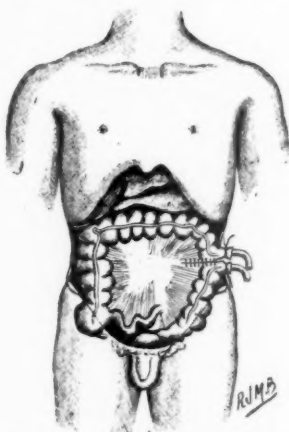
(6) Close the ends of the wound with a few silkworm-gut sutures passing through all the layers of the abdominal wall. No others are necessary.

"When the operation is performed in this way, all the vessels except those in the primary incision are tied before they are cut, and the intraperitoneal work is rendered quite bloodless. The second stage of the operation, that of breaking down the spur with an enterotome, should generally be undertaken about three weeks later. As soon as this has been satisfactorily accomplished, the artificial anus is closed by separating the rosette of mucous membrane from the skin, turning it in, and bringing together the freshened edges of the latter over it."

Such was Paul's operation. It needs to be emphasized that Paul from the very first

advocated wide removal of bowel, practised immediate removal of the affected portion of gut, carefully described a safe method of making a spur, rejected the risky method of resection and suture of the artificial anus, and provided immediate drainage of distended gut. In all these points he was a pioneer. If we remember these details many of the criticisms of the extra-abdominal operation fall to the ground.

Paul's operation, though his account of it seems clear, rational, and successful, did not become well known outside England for some years. This was strange, for surgeons were eagerly seeking for a safer technique, especially since the further experiences of Czerny had shown that a 50% mortality attended resection with suture. It was about three years after Paul had published his paper that Mikulicz-Radecki began to practise resection of the colon by an extra-abdominal method, employing a technique similar to that of Paul, except that at first he did not immediately cut away the gut which had been brought outside the abdomen. In 1902 Mikulicz related his experiences to a Congress of German surgeons and rightly gave credit to Bloch for being the first to recommend external or extra-abdominal resection of the colon by a several-staged operation; for himself he only made the



Preparation of the bowel for the subsequent safe removal of the spur.

[From Paul's paper on "Colectomy" published in the *Liverpool Medico-Chirurgical Journal*, 1895, 15, 382.]

modest claim that he had extended the scope of the operation by a more extensive removal of mesentery with contained glands. Nevertheless he did not excise the artificial anus like Bloch but recommended the crushing of the spur as advised by Paul. Mikulicz knew much of the pioneer work being done in England at this time for in his paper he mentioned the contributions of Herbert Allingham and Walter Edmunds to the subject. Curiously enough he made no reference either to Paul's original paper in 1895 nor his later communication in 1900. The operation recommended by Mikulicz is summarized by him as follows:—

"The primary incision, the enucleation of the tumour, the removal of the lymphatic glands, in short the entire operation is performed exactly as when one operation only is done. If now the tumour has been freed and completely enucleated, it is drawn out of the wound, the loop of gut is stitched to the parietal peritoneum with sutures including only the serous coat, and the abdominal wound is closed leaving only room enough for the loop of the gut. Now only after the abdominal cavity is completely closed, the tumour is excised and an artificial anus established."

At first Mikulicz used to wait twelve to forty-eight hours before he removed the

tumour, but later he removed it at once. A glass tube was fastened into the upper segment of gut and a thick rubber tube fastened to the glass one so that the intestinal contents could flow off. After two to three weeks the spur was crushed and later still the faecal fistula was closed by suture.

Mikulicz showed that by the two-stage operation he had reduced his mortality for resection of the colon from 42 to 12%. He communicated his results to the German surgeons and in the following year went to America and made known his experiences. There can be little doubt that it was due to his name and fame and advocacy that the extra-abdominal operation became popularized and adopted by surgeons generally.

Since that time the opinion of surgeons has varied considerably both as to the parts of the colon for which the extra-abdominal technique was suitable and regarding the actual technique of the operation.

When considering the views of those who contend that the operation is only suitable for mobile segments of the colon, and the statements of others who claim to have extended its applicability by mobilizing the colon, one should remember that both Paul and Mikulicz used the extra-abdominal method for all growths of the colon, wherever situated. Bloch at an earlier date had mobilized the colon to free a growth. Later writers have often stated that they reserved the extra-abdominal operation for those parts of the bowel with a long mesentery, or more particularly for the left side of the colon, whilst the right side of the colon has been dealt with more often by a primary resection from the ileum to the middle of the transverse colon, or by removal of this portion of gut after preliminary ileo-transverse-colostomy.

In 1928 Sistrunk wrote: "The operation may be looked on as applicable for resection only of portions of the colon that are mobile or that may be mobilized without too great injury of the blood supply of the loop that is to be left outside." And again: "Occasionally the Mikulicz operation may be used in resecting carcinomas of the ascending colon, but I doubt that the Mikulicz is often indicated in such cases." On the other hand, F. H. Lahey has advocated resection of the right colon by the extra-abdominal method. Finsterer is in favour of a two-stage resection for right-sided colon-cancer, while for cancer of the left side of the colon he recommends caecostomy followed by a resection of the Mikulicz type.

F. W. Rankin wrote against the extra-abdominal operation fifteen years ago, but increasing experience has inclined him to adopt this technique more frequently. In 1924 he wrote: "Theoretically this type of procedure is admirable from the immediate standpoint of operative mortality, but actually there is slight difference in the death-rate from exteriorization and from radical operations done in one stage on the right colon or in two stages on the middle or left colon. The disadvantages which this type of procedure has outweigh its advantages in that it is followed by a higher percentage of recurrences from cancer than the other types of technique, which fact, in the end, defeats the end for which the operation was undertaken."

And again: "The Mikulicz-Bruns type of operation finds its greatest field of usefulness as a palliative measure. Providing no attempt at a gland dissection and bringing a cancerous growth in direct apposition to the cut surface of the abdomen its percentage of recurrence is higher than that of other types of resection. The ease of execution has popularized its employment and end-results are not confirmed by even a casual study of a group of cases. As originally done, the operation is applicable only to growths which may be easily mobilized."

It is quite clear from reading Rankin's account that his criticism would not apply to Paul's original technique. Lately Rankin has advocated and successfully practised an extra-abdominal method of resection which differs from Paul's method chiefly in that the ends of the bowel are not drained at the time of operation.

In Australia Devine strongly supports Paul's operation and has devised a useful modification of technique. In France Gosset states that exteriorization has lost

ground for all kinds of cancer of the colon though in his opinion it remains the best method for sigmoid cancers.

*I think we may conclude that the extra-abdominal method of colonic resection for cancer or other lesion can be carried out successfully on any part of the colon, though on the right side the ease and success of ileo-colic anastomosis gives us an alternative and perhaps preferable method of operation in one or two stages.*

As Paul stated, when one is dealing with undistended and fairly normal gut there is a field for resection and immediate suture, using either "septic" or aseptic technique, but even in these cases extra-abdominal resection can be carried out.

Cases without much distension may be resected without any previous relief of tension, but when the distension is extreme it may be impossible to draw out the bowel without risk, and in these cases it is wise to perform a preliminary cæcostomy or colostomy.

#### VARIATIONS IN TECHNIQUE

Surgeons differ as to whether the bowel should be cut away at once or after waiting for a day or two to allow adhesions to form. From the very first Paul advocated and practised the removal of the growth and drainage of the proximal end at the time of bringing it out at the primary operation. Mikulicz, though in his early cases he waited a while for adhesions to form, soon practised immediate removal after the careful application of a protective dressing.

Paul and Mikulicz both used to drain the proximal end at once. At the present time in America there is a tendency not to permit immediate drainage of the proximal gut. The growth is cut away at once after the application of clamps which are left on till they fall off by their own weight. Rankin has given the name "obstructive resection" to this method and has had excellent results by its use. Dixon uses a similar technique but prevents undue discomfort and distension by letting off gas above the proximal clamp twenty to thirty hours after the operation. Dixon's modification is a silent criticism of the original obstructive technique. I have not seen the necessity of departing from Paul's technique for in dangerously distended cases I perform preliminary cæcostomy, and when there is only moderate distension the proximal cut end of the colon can be drained safely.

*The crushing of the spur* is usually undertaken two or three weeks after the removal of the growth, but some surgeons do not wait so long. If the ends of the bowel have been approximated in the way Paul recommended there is no need to wait, so long as the pressure is gradual and applied over a broad surface. Devine uses his clamp (which has a narrow face and well-bevelled edges) within the first twenty-four hours. The objection made by Finsterer that the crushing of the spur has led not only to dangerous and even fatal bleeding, but even to small-gut fistula in the colostomy opening with secondary inanition or peritonitis, is only understandable on the presumption that Paul's careful instructions as to the double-barrelled arrangement of the ends of the colon must have been neglected or the clamp applied in an awkward manner.

*The clamp* ought to be applied gradually and should destroy the septum by necrosis and not by cutting. Many clamps have been devised for the purpose. One need only mention those of Paul, Steinke, Findlay, Devine, Wakeley, and Oldfield. Some have been devised to distribute the pressure more equally, others to avoid the awkward projections of the handles of the clamp. Arthur Edmunds devised a special form of clamp which he used in resecting intussusceptions in children; the clamp was applied at the same time as the tubes were inserted into the gut-ends, and a groove was made in each tube to allow its insertion. Paul gave his qualified approval to this.

If a clamp is applied to the spur at the first operation the tissues are soft and easily cut through; if applied after a delay the spur is sometimes hard and thicker and in such circumstances Paul thought it unwise to apply the clamp. When the septum is supple all through he regarded it as safe to apply the clamp at any time.

If any ridge remains after the clamp has separated it may be necessary to apply it again.

*Closing the anus.*—Devine brings out as much spare gut as possible at the primary operation so as to commence closure of the opening even before the clamp has separated; he cuts away redundant mucosa at the daily dressing and sometimes leaves only a small fistula to be closed formally. Hartmann and Lahey have at different times recommended leaving out more of the proximal than the distal gut; Lahey crushes the spur but Hartmann used to resect the anus and suture end-to-end.

Paul used to recommend closure of the artificial anus as soon as the spur had been completely destroyed, but there is certainly some advantage in waiting a while until some of the surrounding oedema subsides and the parts become more normal. Conditions vary greatly and each case must be judged on its merits.

The closure of the anus may be a very simple procedure as one might judge by Paul's description of it. Sometimes, however, it may be difficult to effect a secure closure, especially in fat persons in whom it may have been rather difficult to bring out sufficient gut to make the afferent and efferent limbs quite parallel. Some surgeons prefer to expose the coats of the bowel and open the peritoneal cavity in

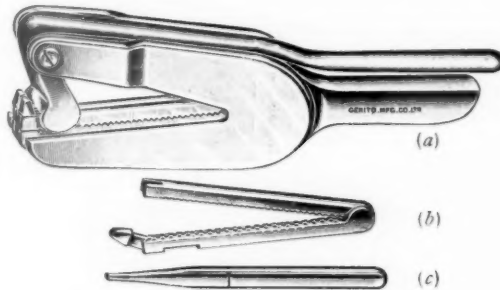


FIG. 1.—(a) Crushing clamp. (b) One (open) blade. (c) Device for opening locked blade.

order to make a complete closure. I have hardly ever found it necessary to open the main peritoneal cavity. Occasionally the closure may be done under local anæsthetic but general anæsthesia is usually preferable.

*Personal experience* has led me to the opinion that for any lesion of the colon accompanied by obstruction there is no procedure so likely to be followed by success as extra-abdominal resection, provided always that extreme distension has been overcome by a previous cæcostomy (or colostomy). It is possible to make a wide resection to satisfy the lymphatic demands shown to be necessary by Jamieson and Dobson's anatomical researches. Any part of the colon except the lower part of the sigmoid may be treated in this manner. For the past seven years I have adopted the extra-abdominal method for all cases of resection of the colon, and in nearly every case I have done the operation with the aid of a few special instruments, which, while not essential to the operation, certainly appear to make it more easy.

After the bowel has been well mobilized through an adequate and suitably placed incision it is brought out of the abdomen and its mesentery ligated in sections at the base of the coil so as to remove lymphatic glands which might be affected. (I always use thread ligatures for the mesentery is fatty and easily retracts.) Care is taken that the base of the coil still has an active blood circulation so that the division of gut will be made without fear of necrosis. The parietal peritoneum is sewn by a few stitches to the afferent and efferent limbs of the coil, and the wound is closed

leaving enough room for the bowel to pass through the opening without being constricted. The bowel is then crushed at the determined places by means of a powerful crushing clamp (fig. 1) with three blades, and divided by the cautery after taking off the middle blade. It will then be found that each end of the bowel can be manipulated easily by grasping the metal blade which occludes it. The two stumps are laid side by side and the contiguous sides of the bowel sewn together by a continuous

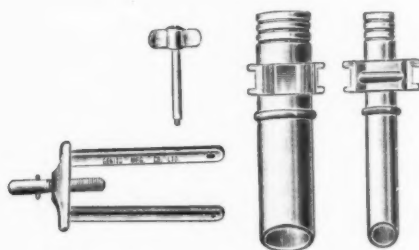


FIG. 2.—Component parts of apparatus for modified Paul's operation. (Two metal tubes, gate and closing screw).

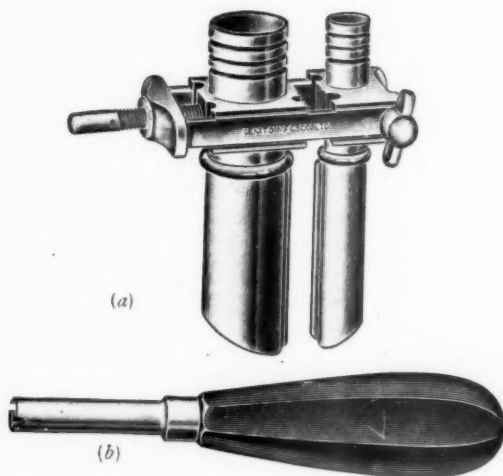


FIG. 3.—(a) Apparatus assembled. (b) Screw-handle.

suture on the side away from the mesentery. The abdominal wound is then protected by rubber tissue or gauze soaked in acriflavine solution before opening the ends and inserting two special tubes. The tubes are made of metal and are of unequal calibre (fig. 2). When dealing with the left side of the colon the larger tube is put into the proximal end of the bowel, but when removing the end of the ileum and the right side of the colon the smaller tube is inserted into the proximal end of the ileum and the larger into the distal colonic end. The tubes are fixed into position by strong

thread, silk, or catgut, which is stitched round the end of the bowel and tied tightly into the groove made for it on the metal tube. The external ends of the tubes are then fitted into a metal gate by means of which they can be approximated and firmly pressed together by screw-pressure. On one tube is a raised ridge, on the other a linear depression, and they are placed in the gate in such a way that the ridge fits into the depression and brings about linear necrosis gradually. (The gate is so constructed that it is only possible to insert the tubes in one way) (fig. 3). Rubber tubing is attached to the end of the proximal tube. The deep ends of the tube, which are specially made oblique to avoid damage to the gut, should be within the abdominal cavity. I have had one or two cases in which there was so thick a layer of subcutaneous fat that the tubes did not reach within the abdomen; in these cases closure of the artificial anus was not so satisfactory.

Each day after the operation the screw is slightly turned and pressure increased. The tubes generally come away in five or six days, leaving one opening and a divided spur. Ten days or a fortnight later the artificial anus is closed unless œdema and thickening round the wound make it advisable to wait a little longer.

The mucosa is well separated from the underlying tissues and the whole thickness of the bowel wall exposed and turned in by a series of catgut sutures. The muscles of the abdominal wall are then united by separate catgut sutures and the skin brought together. A gauze subcutaneous drain is sometimes put in.

*Results.*—There is fairly general agreement that the immediate mortality of the extra-abdominal operation for resection of the colon is smaller than by any other method. Mikulicz had a mortality of about 12%, which is very good when one considers the poor condition of some of these patients. In 1912 Paul published the results of colonic resection in his private patients from 1901 to 1911. There were 18 cases and the only death from operation was the single case in which he abandoned the extra-abdominal method and performed end-to-end suture. Two of these cases were resections of the right half of the colon.

Mr. Gordon-Taylor, who has had a very extensive experience of the extra-abdominal method and has performed 126 operations with only five deaths, states "there can be no doubt that the Paul-Mikulicz exteriorization methods of colon-resection are those attended with the lowest mortality, but that this technique permits the most radical removal of the lymphatic territory is in my opinion somewhat uncertain".

In 1937 C. F. Dixon reported a series of cases with a mortality of from 8–12%. The results obtained by Rankin, who has had very special experience, are better still and as low as 5% or lower. My own results are not based upon a large enough series to permit dogmatism, but they tally with the experience of others who have done larger numbers of cases. In the last seven years I have performed 22 resections of colon. Twenty were single resections, whilst in the other two cases I had also to resect small gut. Of the double resections one died of small bowel obstruction which supervened when he was about to leave hospital, and the other, whose illness had in the first instance become evident because of an abscess in the iliac fossa due to leakage from the growth, died of peritonitis several days after a difficult operation for the double resection. Of the 20 single resections two were for volvulus and recovered so that they passed their motions the natural way without any difficulty.

Of the 18 cases of single resection for carcinoma (all done by extra-abdominal method) one died, a week after operation, of acute dilatation of the stomach and another of pulmonary embolus; the rest made a good recovery in spite of the fact that some of them were very feeble and elderly subjects.

*As regards the likelihood of permanent cure,* from what I have seen the results are likely to be as good as by other methods, for one makes as wide a resection as is made by other techniques.

In conclusion I would like to make a comment on the naming of this operation. I am not in favour of attaching the names of persons to particular operations, but if

a personal name is to be given to the extra-abdominal excision of the colon, my remarks may have shown the justice of attaching the name of Paul to an operation which he was the first to advocate and practise in a manner closely resembling that of the modern surgeon.

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## Section of Psychiatry

President—F. L. GOLLA, O.B.E., M.D.

[January 9, 1940]

### Electrically Induced Convulsions

By F. GOLLA, M.B., F.R.C.P., W. GREY WALTER, M.A.Camb.,  
and G. W. T. H. FLEMING, M.R.C.S., D.P.M.

**Mr. W. Grey Walter :** An account of the method of electric convulsion therapy may be found in the writings of Cerletti and Bini [1], Sogliani [2], Fumarola [3], Kalinowsky [4], Fleming, Golla, and Walter [5], and Shepley and McGregor [6].

We know that a maximal electrical stimulus may be applied to a peripheral nerve many thousand times without doing any harm or causing any irreversible change. In this case each stimulus need be only a millisecond or so in duration and the current required to elicit a maximal response is only of the order of a fraction of a milliamperere. When we come to stimulate the brain the conditions are different, partly because the efficiency of any electrodes is bound to be less than when a peripheral nerve is stimulated owing to the less favourable anatomical orientation of the excitable structures in the brain.

When the human cortex is exposed at operation and stimulation is performed under a local anæsthetic, it is usually found that a current of the order of 10 milliamperes is necessary to elicit a maximal response, and moreover the stimulus must be repetitive and prolonged. Now we know from data obtained from electro-encephalography that the spontaneous electrical activity of the cortex is attenuated about a hundredfold by the overlying tissues : scalp, skull, and meninges. This attenuation is due to the ohmic network comprised by these tissues spreading the potentials out over the surface of the head and providing comparatively low resistance circuits for the currents to dissipate themselves in. This effect is a reversible one, that is to say, a current applied from outside the head would suffer a similar dissipation and attenuation, and since we know that a current of about 10 milliamperes is necessary to stimulate the brain directly, we can compute that something of the order of 500 to 1,000 milliamperes will be necessary to stimulate it when electrodes are placed on the surface of the scalp.

Cerletti and Bini found that potentials of the order of 100 volts were required to produce the necessary current, and from their animal experiments they established that with these conditions a shock lasting about one-tenth of a second produced no

visible changes in the brain substance. As they emphasized in their publications, any attempt to apply Ohm's law to organic resistors is futile since the first effect of a strong current is greatly to reduce the resistance, that is,  $I = \frac{E}{R}$   $\therefore R = \frac{E}{I}$ . This effect has two important clinical results: if we plot the distribution of resistances of a number of patients we get a curve which shows that a number of patients have resistances well over 1,000 ohms and that these high-resistance cases tend to form a group distinct from the low-resistance ones. If, however, we measure the fall in resistance due to the passage of the stimulating current, we find that there is a disproportionately large fall in the high-resistance cases, so that their average resistance during and after the shock is very much closer to that of the low-resistance group than we should have expected (fig. 1). Put in another way, the apparent resistance range is from about 400 to 1,600 ohms; the true range during passage of the stimulating current is only from about 400 to about 800 ohms. Now, it is

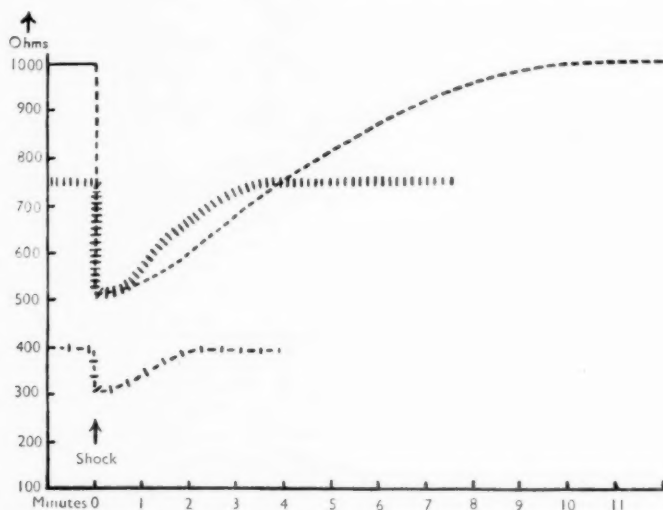


FIG. 1.—Resistance drop and recovery; high, medium and low resistance cases.

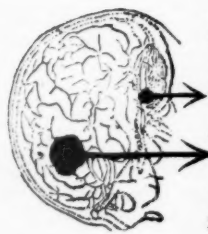
customary before applying the stimulating current to measure the patient's resistance with a low-tension circuit and to adjust the voltage of the stimulating circuit according to the measurement thus obtained. If there were a four to one ratio between the highest and lowest resistances encountered, the highest voltage necessary to produce a maximal response should be four times the lowest voltage; since this latter is about 80 volts it might be thought that voltages up to about 300 would occasionally be necessary. But this is not the case; it has never been found needful to raise the stimulating voltage above 150 volts, which fits in with the observation that the true effective resistance range is only about 400 to 800 ohms. The second application of the resistance fall effect is that if a first stimulus fails to evoke a fit, a second one of the same voltage, encountering a lower resistance, will produce a larger current and will usually succeed where the first failed. The cause of these resistance

fluctuations is not absolutely certain, but in all likelihood is chiefly due to the skin rather than the nervous tissue itself, and for the moment can be neglected except in so far as it affects the setting of the stimulating voltage.

As regards the stimulus itself, there are three variables which should be considered: frequency, wave-form, and duration. The current used by Cerletti and Bini for reasons of convenience is 50-cycle alternating current from the mains, and this choice is entirely to be commended since it greatly simplifies the design of the apparatus. The wave-form, of course, is sinusoidal, and here again a great convenience is achieved since the various calculations can be made according to standard practice. The duration found to be most effective and most certainly harmless is from one-tenth to one-fifth of a second. With the shorter duration each hemisphere of the brain receives five discrete maximal stimuli, since it may be assumed that the effective parts of the wave-train are the half-cycles which are negative with respect to each hemisphere. The alternating character of the stimulus and its short duration are extremely important, and distinguish it from the electrocutory shocks with which it has been ineptly compared. Although stimulation with this type of current is both effective and convenient, it is by no means certain that it is the most efficient possible stimulus for the brain, and it will be interesting to discover whether a stimulus more like the discharges produced by the brain before and during a spontaneous fit would not be even more efficient than the arbitrary commercial product.

The electrodes used for the treatment are very large: those of Cerletti and Bini have an area of about 100 square cm. We have used a simpler design of about a quarter this area applied over the anterior extremity of the frontal lobes. Even with this area of application the whole of the frontal lobes receives an adequate stimulus. The instantaneous effect of applying the current is twofold: firstly a short sharp jerk of every part of the body indicating direct stimulation of the pre-central gyrus, and secondly immediate loss of consciousness. These two facts together indicate the profound and widespread effect of the stimulus. The direct motor jerk is probably comparable to the so-called first myoclonic stage of the cardiazol convulsion, and it is probably this jerk which is responsible for those few accidents to bones and joints which have been reported. The instantaneous loss of consciousness is a valuable feature of the treatment since it is of course associated with complete amnesia. This is both retrograde, covering a few seconds before the stimulus, and antrograde eliminating from the memory events occurring during the early recovery stages. If the stimulus is of subconvulsant strength recovery from the momentary unconsciousness is usually complete in a quarter of a minute, during which time there is usually pallor or flushing, sweating, and sometimes confusion or slight agitation. If the stimulus is just threshold the onset of the convulsion may be delayed up to thirty seconds, but this latent period is usually characterized by a more prolonged period of unconsciousness and more far-reaching vasomotor and respiratory disturbances than when no convulsion ultimately takes place. [The difference between a sub-convulsant and a convulsant dose is usually a matter of only 5 volts. In some patients the threshold seems to be extremely high, in two cases 150 volts had to be applied for a third of a second in order to evoke a fit. Neither of these cases had an unusually high resistance. The complete amnesia for the space of time around the stimulus makes the testing of the patient's threshold particularly easy, since several trial shocks may be administered without causing any sensation, and moreover, when a convulsion is finally produced the further amnesia resulting from this seems to eliminate all traces of the previous testing from the patient's mind.]

With regard to the convulsion itself, it resembles very closely the cardiazol fit or a rather brief spontaneous seizure. The total duration is remarkably constant at about 45 seconds; the tonic phase is rather shorter than in most spontaneous



Record begins about 5 sec. after stimulus.

E.E.G.



E.M.G. Rt. biceps



E.M.G. Rt. triceps



1 sec

E.E.G.



E.M.G.



E.M.G.



1 sec

Higher speed, detail of clonus.

fits and clonic jactitation becomes evident about ten seconds from the onset. Fig. 2 shows simultaneous records of the electro-encephalogram and the electro-myograms of the biceps and triceps of the opposite side. Owing to the huge electric residual artefact from the shock itself it has not been possible to produce a record starting sooner than five seconds after the beginning of the fit; this would be possible only if the latency happened to be unusually long when the record was taken. The tracings in the figure are precisely similar to those which have been obtained during convulsions due to other causes. It is worth noting the nature of the clonic phase, which in the electro-encephalogram is characterized by a polyphasic relaxation oscillation accompanied in the muscles by simultaneous synchronous volleys of action potentials. The two antagonistic muscle groups contract simultaneously during the relaxation phase of the electro-encephalogram and relax together in the crescent phase, the limb falling limply under the influence of gravity. The transition from tonus to clonus is seen as an increasingly frequent and regular interruption of a maximal rigidity.

From the typical character of the fit itself and from its occasionally prolonged latency we may imagine that the effect of the brief but intense and widespread stimulus is to set up in the cortex, or possibly at a lower level, some process similar to that observed in the resting electro-encephalogram of idiopathic epileptics, a process which in favourable circumstances will throw the whole effector system into a state of rapidly alternating rest and maximal activity, a phenomenon which we recognize as an epileptic seizure.]

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**Dr. G. W. T. H. Fleming:** In electrically induced convulsions the shock is followed by an immediate "start" or convulsive movement of all four extremities, facial and spinal musculature. It is only after this initial "start" that the tonic, followed by the clonic discharges, become evident. The initial start reaction has a latent period certainly less than one-tenth of a second. Whether the stimulatory current be sufficiently strong to cause a pronounced fit or be so weak as to give a not greater response than a momentary loss of consciousness, the "start" would appear to be of equal intensity. There is every reason to assume that the "start" is due to the direct stimulation of the motor-cortex by the exciting current. In cases of cardiazol produced fits, instead of a single initial "start" convulsion, a series of such discharges takes place, varying in number from one to ten or more separate convulsions. These are followed by a latent period of varying duration by the tonic and then the clonic phase. The initial phase has been wrongly interpreted as the initial clonus forming part of the fit proper. It is probably the homologue of the electrically conditioned start reaction and due to the primary excitatory action of the drug on the motor cortex. Following the start reaction the clonus and subsequent tonus may develop after the latent period which appears to bear some direct relation to the strength of the stimulating current. These phases, which appear to be identical with those of a true epileptic fit, are of far less intensity than similar discharges

evoked by cardiazol therapy. Possibly the greatest objection to cardiazol therapy is the latent period that elapses between the injection and the beginning of the fit, during which time the patient experiences an emotional state which appears to be intense terror. So horrible does this period appear to be that it is the most potent reason for so many patients insisting on discontinuing the treatment. The electric fit entails a complete amnesia for any events taking place from the moment before the shock is administered till the end of the fit. The strength of the current used would no doubt be extremely painful were it not for the fact that the cerebrum appears to be completely knocked out before integration of the painful stimulus can be effected. Even when a patient receives such a mild stimulus as to cause only the "start" and a purely momentary loss of consciousness, he never has any recollection of the painful stimulus. In many cases the amnesia covers the whole period of adjusting the electrodes. In others it is practically contemporaneous with the shock. The patient never has any knowledge of the fit itself and the period at which he recovers consciousness varies so greatly that it is difficult to assess in any particular case when it is possible for him to appreciate his surroundings. He certainly never complains of any discomfort during the fit. We have for this reason had no cases of patients refusing the treatment of which they know nothing beyond the buckling on of the electrodes. They may, of course, refuse to have this done, in the same spirit as that in which they would decline to have a Riva-Rocci armlet applied.

The commonest difference between the epileptically induced convulsion and that of cardiazol is the very much smaller intensity of the clonic and tonic phases. We never get the intense tonus of the cardiazic patient nor the terrific clonic jerks that may result in dislocation or even fracture of the limbs. The feature that is common to these two artificially produced fits is, as we have said, the initial start reaction, and it is this initial "start" reaction that constitutes the drawback to both forms of treatment. Compression fractures of the vertebræ are practically unknown in idiopathic epilepsy. Fractures are extremely frequent in fits produced by cardiazol and in our series of electrically induced fits we have twice seen them out of a total of 20 cases. We believe that this compression fracture is caused by the sudden violent contraction of the spinal musculature that occurs with the primary motor discharge. It is accompanied by marked flexion of the spine although it occurs in just those spinal segments where the flexion would be most manifest, that is the seventh and eighth dorsal segments. It is probable that the accident has been exceedingly frequent in cardiazol treatment, inasmuch as it is only in those hospitals in which the patients were systematically X-rayed that fractures have been found. It would appear that such fractures may be simply apparent to the patient as a pain of no very great intensity in the back and no more may be heard of them.

There are no recorded cases, as far as we know, of paraplegia or any grave results from such neglected fractures. It is, however, obviously an accident that cannot be neglected. We have no evidence, it is true, as to how often such a condition would give rise to subsequent acute spinal distortion by absorption of the crushed body since it is hardly fair to compare the results of external trauma, in which alone fractured vertebræ have been studied, to those produced by endogenous muscular contraction. An orthopaedic surgeon puts the percentage of cases of fractured vertebræ following falls, such as often occur among jockeys, which give rise to further symptoms, at about 10%. In cases either caused by electricity or cardiazol treatment, it is probably very much less. So far as we know, this liability to fracture of the body is the only contradictory indication to the use of the electrical treatment, whereas in the case of cardiazol fracture of the bones the terror and repugnance of the patient constitute very serious additional drawbacks. As to whether this liability to spinal lesion constitutes a definite contra-indication to the use of the treatment, we cannot pronounce on present results. This must be entirely determined by its therapeutic

efficacy. Schizophrenia causes the death of the nervous system in as large a percentage as an intestinal carcinoma would cause the death of the body.

If convulsion therapy is really of unique value as a curative agent we should not be justified in withholding it on account of these non-lethal dangers any more than we would deny our cancer patients the chance of a major operation. Of the curative effects of these electrically produced fits we would prefer to say nothing at the present moment. Just as with cardiazol, we have seen some marvellous recoveries which the clinician could only attribute to the treatment itself, but the time has not yet come to assess the statistical value.

In conclusion there is a rather interesting feature of the post-epileptic's behaviour which may prove to give some indication as to the mechanism of the curative action of the fits should it exist. Every patient has his own stereotyped manner of behaviour in the post-epileptic stage. Some indulge in rhythmic movements, some always repeat the same utterances, others turn over and go to sleep, others become aggressive or, in rare cases, maniacal. In each the particular type of post-epileptic conduct remains constant until general clinical improvement has become manifest. We have never seen any serious symptoms of respiratory failure or cardiac distress.

The necessary apparatus can be cheaply constructed, and the running expenses are nil compared to the use of expensive drugs. Unlike insulin the electrical treatment does not call for technical skill on the part of the assistant staff.

## The Clinical Applications of Electrically Induced Convulsions

By W. H. SHEPLEY, M.B., D.P.M., and J. S. MCGREGOR, M.B.

OUR apparatus and technique are modifications of those used by Cerletti and Bini (1938) and similar in description to those described by Fleming, Golla and Walter (1939). The apparatus is wired in two circuits, one a low-voltage direct current circuit for measurement of the patient's head resistance, the other an alternating current circuit, giving a voltage which can be varied between 50-150 volts. The low-voltage circuit gives a current of one milliamperere. To sensitive patients even this small current can be unpleasant if applied suddenly; therefore it is gradually applied to the head by means of a potentiometer whereby no complaint of tinglings or other sensations arises. The resistance of the head is read on a scale round the knob of the potentiometer. Another feature is that the low-voltage circuit can be made independent of mains fluctuation, a fluctuation in the order of from 6-7%, avoidable by means of a standard resistance which can be inserted into the circuit. In this way the feeding voltage of the potentiometer is kept constant, and the obtained resistance values are not subject to any fluctuations. The time during which the current is allowed to flow is predetermined on a time-switch of the condenser type. This gives readings of 0.1 to 0.5 of a second. The current flowing through the head is measured in milliamperere-seconds on a meter employing the ballistic principle so as to permit the reading to be taken when the needle reaches the maximum of its excursion. The reading, divided by the time, gives the current in milliamperes. The electrodes consist of silver-plated strips of flexible copper, mounted on rubber cushions fixed to an adjustable clamp. The area of the electrodes is about 38 sq. cm.; therefore the current density at any one point is never very high, being less than the average current recommended for use in diathermy. Before application to the head the electrodes are covered with linen which is soaked in a 20% solution of NaCl.

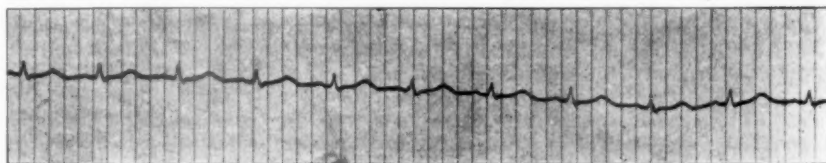
They are applied to the head at the junction of the parietal and temporal bones, over which site a contact paste has been smeared. This ensures good contact between the electrodes and the scalp and helps to ensure that conduction will not take place to parts other than the head. It is interesting to note that if the chin or head is touched a shock may be felt, but not from any other part of the body. It appears to us that the time during which the current is allowed to flow is a more important factor in the induction of a fit than is voltage, for we have frequently been able to obtain a major fit by increasing the time when an increase in voltage had proved ineffective. The reason for this appears to be that when time is increased the number of stimulations reaching the cortex increases proportionately. Nevertheless we hold the view that the fit might be induced either by a high voltage for a short time, or by a low voltage for a longer time. As to which of these methods is the more desirable is a question to be decided by physiological experiment, preferably other than in man. The voltage ordinarily used by us ranges from 100–120 volts, the time 0.2 second. The effect of the passage of the current is to induce either a major fit or merely transitory loss of consciousness—the so-called abortive fit. When no fit is obtained by the first shock, there appears to be no contra-indication to reapplying the electrodes and making another attempt. When this is done it is observed that there is a fall in the head resistance. This fall appears to be progressive following each successive shock until a minimum in the region of 100–200 ohms is reached. The reason for this is not clear, but it is possible that it is the result of tissue electrolysis. We have in the case of one patient made six successive shocks before the major fit was obtained.) There was no protest from the patient and so far as could be observed no ill-effects. In the major fit evacuation

TABLE SHOWING EFFECTIVE INDUCTION OF FIT BY INCREASING TIME.

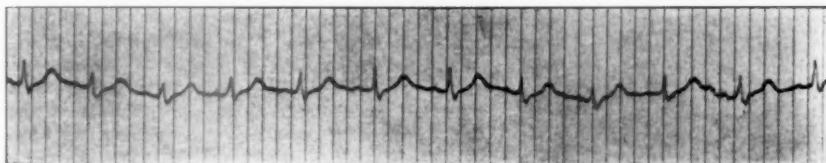
Date	Time	Resistance	Volts	Milliamps	Fit
2.12.39	0.1 sec.	750	85	450	—
2.12.39	0.1 sec.	550	95	600	—
2.12.39	0.1 sec.	420	120	900	—
5.12.39	0.15 sec.	500	120	850	—
5.12.39	0.20 sec.	250	120	900	+

of urine or faeces has been observed, as also has seminal emission. The effect of an abortive shock upon the patient varies considerably, from momentary loss of consciousness to generalized tonic spasm of muscles with slight opisthotonus and opening of the mouth, though the further stages of a major fit do not follow. It would appear that anticonvulsant drugs such as paraldehyde, have the effect of increasing the patient's resistance to the current. One of our patients who had 3 drachms of paraldehyde, together with 30 gr. of sulphonal the night before treatment, required a much higher current than usual before a major fit could be induced, and a similar result was noted on subsequent occasions. Another patient was given one capsule of epanutin for three days before treatment and the voltage had to be increased by 10 volts to induce the major fit. This patient had previously shown major convulsion consistently with the same voltage and time. This compares with the observation of Cook (1938) that a larger dose of cardiazol was necessary when such drugs had been administered. [The effect of application of the current on the heart has been observed, and it appears that at the moment of passage of the current the heart stops; the exact duration of this pause cannot be estimated, for almost at the same moment there is some movement on the part of the patient so that further observation either of the pulse or cardiac sounds is rendered impracticable, and it is of course impossible to take an electrocardiogram during the passage of the current.] Electrocardiographic records have been made in some patients, both before and after the shock, and it is

of interest to note that there is an increase in the amplitude of the ventricular complex of the order of 0.2 of a millivolt in records taken three minutes after the shock. Indeed, in the flaccid stage immediately after the fit, this increase was so great that the beam of the cathode ray of the electrocardiogram left the fluorescent screen. While the reason for this is not clear, it may be, as suggested by Fleming, Golla, and Walter (1939) in the case of the electro-encephalogram, that not only the head but also the body becomes charged in the nature of a condenser. We have applied the method to patients in insulin coma, without untoward result. In these cases we take the precaution to see that patients in the so-called wet coma are sponged dry. The normal dry skin is a poor conductor of electricity and MacDonald Critchley (1936) quotes that the dry, horny palm of a workman has a resistance of from 1 to 2 million ohms. Notwithstanding, under circumstances favourable to the passage of the current, comparatively small voltages, in the region of 100, have proved fatal. It is well known that many electrical accidents occur in the bathroom when a short-



E.C.G.—Lead I. Before shock.



E.C.G.—Lead I. Three minutes after fit, showing increased amplitude of Q.R.S.

circuited switch has been touched by a wet hand. We therefore see to it that pillows and bed linen, if moist with perspiration, are changed. With non-insulin patients the major fit most commonly occurs immediately, or within a few seconds, though we have noted a delay of thirty seconds in several cases. A delay of thirty seconds in coma patients is, however, common, or even as long as one and a half minutes. The reason for this is probably that cortical activity depressed by the action of insulin takes a much longer time to respond to the number of stimuli applied to it by the alternating current. Commonly, too, the fit is atypical. One of our cases had on two occasions a fit almost confined to the left side of the body. This began with clonus of the circumoral muscles and spread to involve the left arm and leg in clonus. There was no obvious tonic stage. The pupils, commonly small and occasionally fixed before the application of the current, dilate fully, and the light reflex is lost. Some cases immediately after the shock become slightly cyanosed and this deepens until the face becomes almost purple, and then the major fit occurs after a delay of about fifty seconds. [An interesting feature of the application in insulin coma

is that when several shocks have been given without the induction of a major fit, there is a tendency for the patient to come out of coma, and this is reflected in the blood-sugar which shows an increase of about 15 mgm.%. We have not chosen any particular time after the onset of coma, for application of the shocks, though Georgi (1937), who introduced the summated method of treatment with cardiazol, suggested that it should be done from one to two hours after the onset of coma. The time selected and the depth of coma appear to make little difference to the results of application of the shock. It has also been noted that after the shock, bile, absent in the gastric juice before the shock, now becomes present in fairly large amounts. It has been suggested by Cerletti and Bini, also by Kalinowsky (1939) and others, that the area of the brain stimulated is the Vogt-Brodman area 6a $\beta$ . While this area certainly seems to be the one mainly involved, the presence of bile in the gastric juice after a shock, even when no fit is induced, and the frequent development of a dusky cyanosis of the face after an abortive shock, suggests vagal activity consequent upon stimulation of the medulla.

Our 50 cases already treated by the electrical method were for the greater part cases of long standing which had failed to respond to cardiazol treatment, others were drug-convulsant cases now continued by electrical means. From this limited number, admittedly too small for any conclusions to be drawn, certain indications appear. The cases which had previously failed to respond to cardiazol, equally have shown no response to electrical treatment, nor does it appear likely that they will. Cases which had previously shown response to cardiazol or triazol, equally appear to respond to electrical treatment. These include cases of katatonic schizophrenia, schizophrenic reactions of exogenous type, one a recurrent post-puerperal katatonic stupor, pregnancy twice having appeared in causal relationship, also certain depressives including agitated melancholias, together with cases of paraphrenia, recurrent mania, and hysteria. An interesting group consists of five patients who had previously responded to cardiazol but had consistently relapsed if treatment was withheld for longer than a fortnight. These patients appear to require a "maintenance" treatment to prevent certain relapse. Hitherto this matter of indefinite continuance has presented a difficult problem because of the dread of treatment, especially of a treatment without apparent end-point. These five individuals approximate to the katatonic schizophrenic type without appreciable mental deterioration, so that they attain normality apart from their psychotic phases in which dyskinesia is an outstanding feature. They have been found to respond equally to electrical treatment without the accompaniment of fear. [This feature of "maintenance" treatment by the new method appears to open up interesting possibilities for out-patients' clinic application, since provided the patient is willing to attend for periodic treatment, resumption of ordinary outside life appears possible. This possibility is furthered in two directions, namely, the removal of fear which otherwise would render voluntary attendance beyond expectation, and the absence of "after-symptoms", such as vomiting, confusion, and excitement, which have hitherto required confinement to bed for an hour or more and a close nursing supervision. That the therapeutic results of the method described must stand or fall with those of shock therapy in general, we are agreed. The indications and contra-indications appear essentially similar, as also is the preparation of the patient. In furtherance of Dr. Skottowe's "Plea for Proportion in Psychiatry", where shock therapy is concerned, we would attempt some review of apparent indications and contra-indications as they appear to us. Upon a wider basis of clinical experience of shock therapy in general, including 250 cardiazol treated cases and 50 treated by insulin, it appears possible to indicate broadly certain cases which do not respond and those in which treatment appears likely to be beneficial. It is generally admitted that for the most part chronicity is of unfavourable import where shock treatment is considered. Those cases which

tend to recover spontaneously may recover more speedily given shock treatment. What to-day is described under the term of the Schizophrenias is a group so wide as to be in danger of losing precise meaning. It appears fundamentally important to differentiate between the true endogenous malignant schizophrenias and the relatively benign exogenous reaction types, many of which are recoverable. Among the shock-resistant cases we find the true endogenous schizophrenias, well termed dementia præcox by the older clinicians. These occur in individuals of pronounced constitutional inferiority with poverty of organic endowment. They are marked by physical signs of cardiovascular hypoplasia and hypofunction, together with general asthenia, poorness of sex differentiation, and consequent physical tendencies towards those features, the presence or absence of which characterize the opposite sex. These circulatory and endocrine features were well described by Lewis (1923). Such individuals morphologically tend towards the asthenico-athletic or less commonly the dysplastic types of Kretschmer, but morphological considerations alone are not sufficient to differentiate them, since function and capacity to react are more important than structure. Physical signs such as persistently cold, blue extremities even in warm weather, denote poorness of peripheral circulation and inherent lack of vitality. Examination by orthodiagraphy shows smallness of the internal viscera, in particular the heart and aorta, the latter tending towards smallness of calibre. Such findings are confirmed by post-mortem examination and are characteristic of dementia præcox, which *per se* is a sufficient certifiable cause of death. Mentally such individuals show emotional poverty and apathy, the negative aspects of mentation outweighing the positive. In such individuals the prognosis from the first appears unfavourable, and in their varying degrees they form the larger proportion of our chronic mental hospital population. In our experience they are quite unresponsive to shock therapy, appearing to lack the essential capacity to react. A further group which appears therapeutically resistant consists of the fixed delusional classes whether paranoid dementia, paraphrenia, or paranoia. Of these, however, the two former may at least be relieved of hostility and be rendered more accessible and sociable. There is nevertheless at least one exception to the above generalization; that is, where the delusion is the direct outcome of altered feeling-tone such as occurs in certain depersonalization states of depression: here, convulsion therapy has proved capable of removing even a fixed delusion. Such was the case where a female patient steadfastly declared for a period of months that she was made of wood. This delusion speedily disappeared following cardiazol exhibition. True manic-depressives of affective cyclothymic type characterized by periodic elation and depression are definitely rare in mental hospitals, but when they occur, they approximate to Kretschmer's pyknic type as regards habitus. Like the schizophrenias, they appear to occur in both endogenous and exogenous forms. Irrespective of causation these cases are essentially reactive, in the sense that sensory stimulation results in ready motor response; and in such, cumulative inhibition does not readily occur. Clinically they do not show the features and extremes of inhibition and excitement in marked degree, but rather phases of irritability and agitation, or alternatively a quiet melancholy or mildly demonstrative elation. The term "manic-depressive", although properly applied to these, appears nevertheless unfortunate, since the term suggests the more obvious extremes of excitement and inhibitory depression such as characterize the dyskinetic schizophrenes with all-too-frequent confusion. As regards convulsion therapy, these affective cyclothymes tend to react badly, not infrequently showing pallor, rapid feeble pulse, and signs of physiological shock. Such signs appear accountable to their relatively large splanchnic area in these individuals of pyknic body type. Essentially they are not of cortical inhibitory type and in them convulsion treatment appears generally to be contra-indicated. To turn now to indications for convulsion treatment in particular, as apart from

shock therapy in general, which of course includes insulin, a review of our successes makes clear especially that those cases which can be grouped as "inhibitory" in the physiological sense are of favourable import: it is a group largely composed of dyskinetic schizophrenes. These include cases of stupor, both depressive and katatonic, and a wider group of what may be termed schizoid depressives, individuals with less marked motor manifestations, but whose depression is characterized by peculiar persistency and malignancy of quality. That the excitatory counter-phase of katatonic stupor, and depression, should also respond, though less conspicuously, seems understandable as furthering a "release" phenomenon already begun. Whilst the above appear to belong to the same constitutional type, for the most part asthenico-athletic schizophrenes of good organic endowment, they may show not only alternations of phase, with or without intervening normality, but also predilection towards either extreme, inhibitory or excitatory. There are certain individuals in whom phases of pseudo-maniacal excitement marked by hypermotility recur every few weeks, but in whom no depressive phase appears. In such the intervening normality represents no more than that degree of inhibition which is the proper function of cortical control. To this condition the name recurrent mania is commonly misapplied but it is important to observe that true elation is not a feature nor indeed is any sustained affectivity. A seeming silliness of behaviour is however characteristic, and is resultant from disharmony between affect and ideation. The above described clinical picture clearly aligns these cases with the schizophrenias. They appear indeed to represent truly, acute schizophrenic episodes albeit of short duration; episodes interrupting normality and not featuring a continuous psychosis. Moreover they are seemingly constitutionally predisposed and endogenously determined. In such individuals this excitatory phase can be anticipated by induced convulsions towards the end of normality, or can be curtailed by such, on first appearance of relapse. There appears in short to be not only a broad basis of selection for treatment, but more precise indications regarding time of application and frequency of application relative to alternation of phase and the individual's response to treatment. We are conscious of the incompleteness of this brief survey, but submit that a study of basic constitution is an essential guide to case selection where shock therapy is considered, if such is to be more than a merely empirical procedure.

#### CONCLUSION

To revert to electrically induced convulsion therapy as compared with other methods, the chief points can be summarized as follows: The electrical method is well tolerated by the patient who is freed from the dread which hitherto was associated with these necessarily continued treatments. The disagreeable sequelæ of drug-convulsant treatment are notably absent, such features as vomiting, confusion, and psychomotor restlessness which formerly required close "after-supervision" are not evident. The method by its nature is devoid of such former technical complications as thrombosis of veins, a feature of a special importance where insulin is used either subsequently or in combination. The method can readily be combined with other treatments such as insulin, and being a physical therapy, removes all question of toxicity or cumulative action. In virtue of the last-mentioned facts, the procedure is completely controllable, a succession of fits not intended being unknown. The method is ideally suitable for the indefinitely prolonged maintenance treatment in those not-infrequent cases where an occasional convulsion is essential to prevent relapse. The method may well reduce the incidence of fracture since our impression is that the fits upon the whole appear less strong. In our series of some 200 induced major fits neither fracture nor dislocation has so far occurred. The method offers of ready repetition without the attendant difficulties of a struggling patient, and indeed the abortive fit induces quiescence and a ready acquiescence to further

treatment, in marked contrast to cardiazol administration under similar circumstances. From the administrative point of view the method offers advantages of economy, also less nursing attention and supervision.

It remains for us to thank Dr. Rees, Medical Superintendent of Warlingham Park Hospital, for suggesting the electrical treatment, and by his good offices making such possible.

To Dr. Kalinowsky we are indebted for practical aid and advice, freely given.

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*Discussion.*—Dr. L. KALINOWSKY: The first experiences with electric convulsion treatment (E.C.T.) in this country are of recent date. Professor Cerletti sent me a report on the two years' work done at his clinic in Rome.

The time of observation is still too short for a definite judgment. Figures of the immediate results have been given from a first series of 3,000 fits in more than 100 patients. In those cases where the duration of the psychosis was less than six months complete recovery was obtained in 80%; the remaining 20% were much improved. The complete recoveries decrease, as in all the other shock treatments, with the increasing duration of the disease. In cases of one to three years' duration only a third showed recovery or was much improved. Still older cases showed improvement in 50% but never complete recovery.

As to the number of fits, the optimal effect was obtained in the first group of recent disease after 15 complete and four incomplete fits on an average.

I have not seen any striking difference in the quality of the results between the various groups of schizophrenia. From the beginning we have seen most surprising reactions in the depressive group of manic-depressive psychosis. Cerletti now regards his results in these cases as still more brilliant than in the treated schizophrenics.

Here are some results of early investigations: Accornero found a rise of systolic blood-pressure immediately after the fit by 50 to 60 mm.Hg and a fall to normal within thirty minutes. The diastolic pressure does not rise up to more than 10 to 15 mm.; in other words the pulse pressure is considerably increased. These figures might be of still greater interest in problems of epilepsy than those from cardiazol fits.

The effect on blood-sugar was a rise by about 20% with a return to normal within two hours, but only when unconsciousness and not a real fit was obtained. Fractures and dislocations have never occurred, probably because the fit seizes an already unconscious and relaxed patient and not the severely struggling patient after cardiazol injection. Cerebral complications or mental sequelæ have never been seen during or after the treatment within the two years' application of the method. That there are no anatomical reasons to expect such complications is shown by Cerletti and Bini by means of extensive investigations on shock-treated animals. They found irreversible cell lesions in insulin-treated as well as in cardiazol-treated animals. But they never found any in dogs treated with E.C.T.; here only reversible changes such as Nissl's acute cell disease were seen.

I think that the E.C.T. has proved its harmlessness by the fact that in Italy where the method is used in all mental institutions an enormous number of fits had been produced without any incident. This aim has been achieved by means of a standard apparatus introduced in England on the

suggestion of Dr. Rees by the "Solus" Electrical. Cerletti and Bini had an apparatus in mind which, without special knowledge of electrophysiology, can be employed with the same reliability by every doctor of a mental institution. In order to produce the fit with a minimum of voltage they constructed an often modified electrode which enables them to apply a very strong local pressure without inconvenience for the patient. In this way they get a very low resistance and obtain fits with an average of 80 to 100 volts passing 0.1 sec. This short time limit is recommended by Cerletti because animals can stand extremely high voltage but are endangered after a very long time of current passage by the long tetanic state of the respiratory muscles.

It was due to those technical precautions that the method could prove its harmlessness and might therefore become a progress in modern shock therapy.

Dr. DONALD BLAIR: During the early days of cardiazol and insulin therapy I noted similar phenomena to these described by Dr. Fleming, and published a paper on the subject in the *Journal of Mental Science*, September 1938. I then noted the following types of movements to occur either during the recovery phase following the fit, or if an insufficient dose to produce a fit was given:—

- (1) Myoclonic twitches.
- (2) Various chewing, sucking, and munching movements of the mouth; pouting; grimacing; and facial contortions.
- (3) Various twistings and writhings.
- (4) Psychosomatic restlessness.
- (5) Some patients remained quiet apart from the actual phenomena of the convulsion.

I have been unable to correlate such occurrences with any particular mental symptoms or types of psychosis, and fail to see how Dr. Fleming is to explain the therapeutic effects of electric convulsion therapy on such lines.

I was disappointed to hear from Dr. Fleming of the two cases of fractured vertebræ in a comparatively small number of convulsions. In the early days of cardiazol therapy major complications were considered extremely rare; such an early record of fractures in electric therapy must put one on one's guard against excessive enthusiasm.

## Section of Therapeutics and Pharmacology

President—Sir WILLIAM WILLCOX, K.C.I.E., C.B., C.M.G., M.D., F.R.C.P.Lond.

[December 12, 1939]

### Preliminary Observations on the Use of Convalescent Serum in the Treatment of Acute Rheumatism

By C. A. GREEN, A. J. GLAZEBROOK, S. THOMSON, and W. A. HOPKINS

#### INTRODUCTION

IN certain communities of young, male adults engaged in strenuous physical training, there has been a considerable amount of acute rheumatism. In a few individuals there was a definite history of some manifestation of rheumatism before admission to the training establishment, but in many the first attack was experienced within a few weeks of entry. The mode of onset was extremely varied. In the majority of cases, nasopharyngeal infection antedated the insidious onset of stiffness and pain in one or more joints which demanded medical attention, and the patient was then found to be febrile. In others, the disease appeared with dramatic onset of hyperpyrexia, sweating and polyarthritides, as in typical rheumatic fever. On the whole, the joint lesions in this latter group of cases have not been striking. Large effusions were not common, but tenderness, slight puffiness and oedema of surrounding tissues, were the usual manifestations. Flitting in character, the joint lesions rapidly improved and left no apparent permanent injury. The initial pyrexia rarely lasted longer than one week irrespective of the treatment adopted, but exceptional cases continued febrile for months. Remissions and relapses occurred frequently and symptoms were greatly relieved by salicylate therapy. Despite the apparent mildness of infection in the early stages of the disease, the cardiac complications have been all too frequent, and have occasioned great anxiety on account of the resulting incapacity. Fortunately, a number of patients did not show any tendency to develop cardiac lesions. As these were otherwise healthy young adults, it was decided that serum, taken from such patients during convalescence, should be tried in the more acute phases of the disease in subsequent cases.

#### METHODS

*Preparation of serum.*—Patients who were in good general condition about the fourth to eighth week after the temperature had settled were selected for the supply of serum. Other criteria of suitability included the absence of all indications of

cardiac complications, and the approach of the erythrocyte sedimentation rate to normal limits. From each patient, 300 c.c. to 400 c.c. blood were obtained, the serum separated, filtered, and preserved by the addition of 0.3% phenol. In this preliminary investigation, the serum from each patient was subjected to the Wassermann reaction and, if negative, was ampouled. Routine sterility tests were performed on each batch of serum.

*Dosage.*—The serum was given either intramuscularly or intravenously in doses of 10 to 20 c.c. Exceptionally larger doses were used as detailed in individual case-notes.

*Additional therapy.*—As far as possible, no form of treatment other than general nursing and local therapy was used in conjunction with serum. Depending on the preference of the clinician in charge of the case, additional measures such as the use of salicylates, supplemented serum therapy.

### RESULTS

Serum has now been used in the treatment of 15 patients divided into two groups : those treated by serum alone, and the remainder in whom serum therapy supplemented the action of other modes of treatment such as salicylates.

*Serum without salicylates.*—In 10 cases, no salicylates or allied preparations were used throughout the illness. Of these, seven cases, which included six primary attacks and one recurrence, reacted favourably to the treatment. Details of two of these successfully treated cases were as follows :—

*Case 1.*—G. M. (MX. 57749), aged 15 years.

*Family history.*—Mother, father, and two siblings—alive and well with no history *re* rheumatism.

*Previous history.*—Tonsillectomy at 8 years—no rheumatism.

*Present history.*—21.8.38 : Joined training establishment.

8.11.38 : Common cold.

30.11.38 : (Chart I) Admitted to hospital, complaining of joint pains in arms and legs. The knee-joints were both swollen, extremely painful and, in the right knee, there was a considerable effusion. The condition remained unchanged for next five days in absence of any therapy other than careful nursing and local treatment.

6.1.39 to 9.1.39 : Intramuscular injection of 10 c.c. convalescent serum given daily. The pains in the joints were greatly eased within a few hours of the first injection, and the patient

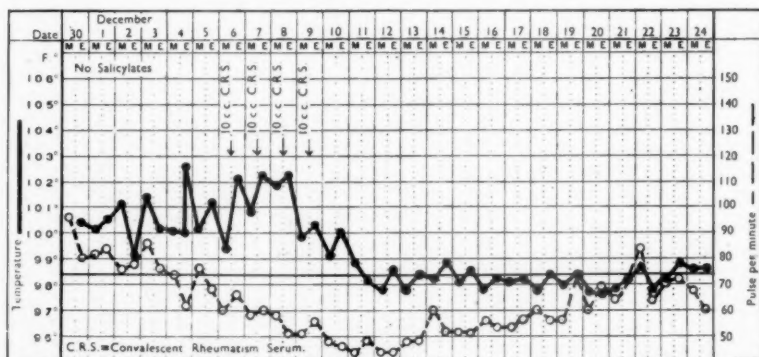


CHART I. CASE 1.

passed his first good night on the sixth day of illness. There were no further joint lesions after the ninth day, and the temperature had returned to normal within six days of serum being first given. A mitral systolic murmur was noted on the fifth day of disease but disappeared during the fifth week. Apart from marked bradycardia during the second week, the remainder of convalescence was uneventful. There was no relapse, no evidence of valvular disease on discharge, and the patient has been on duty for several months in apparent good health.

*Case 2.*—K. A. (JX. 162936), aged 15 years.

*Family history.*—Nothing relevant.

*Previous history.*—No rheumatism—tonsillitis infrequent.

*Present history.*—29.8.39: Admitted to hospital with complaint of pain and swelling of left ankle and, later, right ankle. The right knee was definitely swollen, very tender, and both ankle-joints were stiff and sore. Condition remained unchanged on following two days.

31.8.39 to 2.9.39: Intramuscular injection of 10 c.c. convalescent serum given daily. The temperature and pulse-rate progressively fell during next four days and pain was relieved within twenty-four hours. Convalescence was uneventful without any relapse. Finally returned to duty without evidence of cardiac damage.

The five other cases were of this same type: convalescent serum, given in the early stage of the disease, was followed by a rapid fall in temperature, and speedy relief of the symptoms. Six of the seven successfully treated cases were primary attacks, and the seventh was a recurrence. No case has relapsed since treatment and all have returned to duty.

Details of the three cases of this group which did *not* react favourably to serum therapy were as follows:—

*Case 8.*—W. E. (3148/39), aged 27 years.

*Family history.*—Mother had rheumatic fever and died from rheumatic carditis. Father and two siblings alive and well.

*Previous history.*—Fleeting joint pains for nine months previous to present attack.

*Present history.*—20.3.39: Joined training establishment.

9.5.39: Admitted to hospital complaining of pain in both knees. The left knee was definitely swollen, warm, and very tender. On the same day, 20 c.c. convalescent serum were given intramuscularly.

10.5.39: Knee-joint still swollen and temperature rising.

11.5.39: Knee little easier, but left ankle now affected.

12.5.39: Joint pains stopped but temperature still 99° F.

Remainder of convalescence was uneventful. There was no cardiac complication and patient was discharged to duty.

*Comment.*—In the absence of any effect on temperature and on account of extension to left ankle-joint, serum had no effect in this case. Only one dose was given and continued treatment might have been more successful.

*Case 9.*—A. M. (3136/39), aged 18 years.

*Family history.*—Nothing relevant to rheumatic infection.

*Previous history.*—No previous diseases.

*Present history.*—8.3.39: Admitted with complaint of pain in soles of feet and later in knees. Pain, swelling, and tenderness of both wrists. 20 c.c. convalescent serum given intramuscularly.

9.3.39 to 11.3.39: Temperature continued to rise, and joints remained swollen.

12.3.39 to 13.3.39: Temperature fell and joints improved.

14.3.39: Intramuscular injection of 10 c.c. serum.

15.3.39: Symptom-free, and joints apparently normal.

The remainder of convalescence was uneventful, despite the slow return to normal limits of the erythrocyte sedimentation rate and formol-gel reaction. There were no cardiac complications and the patient returned to duty.

*Comment.*—The first injection of serum had no effect in arresting the disease, and although the second injection was followed by amelioration of symptoms, the chart (not illustrated) indicated that this was a chance association. However, it was noted that there were no further joint manifestations and no cardiac lesions, although the sedimentation rate remained abnormal for a further three weeks.

*Case 10.*—A. M. (1840/F.T.), aged 16 years.

*Family history.*—Nothing relevant to rheumatism.

*Previous history.*—Measles, rubella, no rheumatic manifestations.

*Present history.*—10.1.39: Joined training establishment.

14.2.39: Common cold and tonsillitis.

14.3.39: Complained of generalized joint pains and abdominal pain.

17.3.39 to 21.3.39: (Chart II) Intramuscular injection of 10 c.c. convalescent serum given daily. Joint pains ceased 20.3.39, but temperature remained elevated.

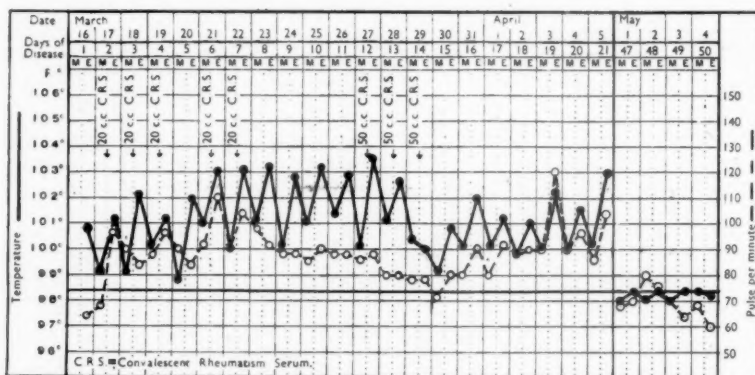


CHART II. CASE 10.

24.3.39: Pericarditis.

27.3.39 to 29.3.39: Intravenous injection of 50 c.c. convalescent serum daily.

30.3.39: Temperature dropped to 99° F., and patient much easier. Stock of serum exhausted, and temperature again started to rise.

5.4.39: Massive pericardial effusion and also effusion at left base.

6.4.39: Dyspnoea very distressing, and pericardial effusion tapped to relieve pressure.

7.4.39: Condition unchanged, and tapping repeated.

11.4.39: Pleurisy at right base, and consolidation at left base, pericardial effusion decreasing.

He made a steady recovery, the pneumonitis and pericarditis clearing up. A blowing, mitral systolic murmur was heard when the pericardial rub ceased, but this finally disappeared, and he was discharged to duty looking extremely fit. He has now been on duty without relapse.

Two blood cultures during the pyrexial period, and both pericardial and pleural effusions were sterile.

*Comment.*—Despite the failure of the initial course of serum to prevent the onset of pericarditis, the clinician was of the opinion that the large doses of the second course had a beneficial effect on the complication. Unfortunately, the stock of serum was exhausted. The temperature chart also suggested that the process was temporarily arrested by the serum therapy.

In this group of ten cases, nine have now been at duty for several months without relapse, and with no evidence of permanent cardiac damage. The remaining case probably has a valvular lesion, but tolerance has always been good and he is also back on duty.

*Serum and salicylate therapy.*—In five cases serum was used during some stage of treatment in combination with or as an alternative to salicylates.

*Case 11.*—L. W. B. (550788. 6386), aged 19 years.

*Family history.*—Nothing relevant.

*Previous history.*—Rheumatism three years previously.

*Present history.*—Started with pain in both feet. Next day, both knees were affected, and on the following day, both wrists.

5.12.38 : Both wrists, right knee and right foot, swollen, tender, and painful. Pain also in both shoulders and back of neck. Convalescent serum not then available, and salicylate therapy was started. Response satisfactory.

13.12.38 : Left wrist only slightly painful. Temperature continued in normal limits from 8.12.38 and the erythrocyte sedimentation rate and formol-gel reaction settled. Salicylates stopped 31.12.38.

5.1.39 : Left wrist again swollen and painful. Salicylates again checked temperature. Convalescence was uninterrupted for next thirty days, but on 16.2.39 sedimentation rate and formol-gel reaction rose for no apparent reason.

24.2.39 : Relapse with pyrexia (T. 106.1° F.), pain, swelling, and tenderness of right elbow, right wrist, and left knee. 10 c.c. convalescent serum given intramuscularly.

27.2.39 : Temperature normal.

28.2.39 : Salicylates restarted.

Convalescence continued uneventful with no evidence of carditis at any stage, and he was discharged for duty.

*Comment.*—The actual effect of serum on this relapse was obscured by the use of salicylate. However, the temperature had returned to normal and the pains were relieved before salicylate therapy was restarted.

*Case 12.*—F. W. (JX. 161128), aged 15 years.

*Family history.*—Nothing relevant.

*Previous history.*—Tonsillitis six weeks prior to admission.

*Present history.*—16.3.39 : Admitted with pain and stiffness of left arm and shoulder. Salicylate therapy started.

20.3.39 : Temperature normal but clinical evidence of early cardiac involvement.

4.4.39 : Localized mitral systolic and diastolic murmurs confirmed valvular lesion present.

7.4.39 : Erythrocyte sedimentation rate and formol-gel reaction remained abnormal, and both shoulder-joints became active. Responded to further salicylates.

10.4.39 : Symptom-free, but erythrocyte sedimentation rate and formol-gel reaction still high.

2.4.39 : Pain in the region of thoracic vertebræ and over scapulæ, again responded to salicylates. No further joint-pains but continuing high erythrocyte sedimentation rate and formol-gel reaction until 3.6.39 : Right wrist-joint ; 4.6.39 : Right elbow-joint.

1.7.39 : Digitalis on appearance of auricular fibrillation.

5.8.39 : Complaint of pericardial pain and rapid rise in temperature and pulse-rate heralded onset of pericarditis. No salicylates were given at this time, but after six days (10.8.39) 15 c.c. convalescent serum were given intramuscularly.

- 11.8.39: Slight fall in temperature. Intravenous injection of 10 c.c. convalescent serum.  
12.8.39: General condition improved, pulse stronger and more regular. Intravenous injection of 20 c.c. convalescent serum.  
13.8.39: Improvement maintained.  
14.8.39: 20 c.c. convalescent serum intravenously.  
20.8.39: 20 c.c. convalescent serum intravenously. Within two minutes marked dyspnoea and cyanosis, pulse almost imperceptible. Adrenaline and oxygen given; patient rallied in few minutes.  
21.8.39: Felt much improved.  
23.8.39: 20 c.c. convalescent serum intravenously. Again respiratory distress within five minutes, relieved by adrenaline and oxygen in thirty minutes.  
24.8.39 to 7.9.39: Uneventful.

*Comment.*—This case indicated the gravity of the disease and left no doubt as regards the diagnosis. As shown, salicylate therapy alleviated the joint pains but did not prevent the recurrence of arthritic lesions, nor the development of pericarditis. It was interesting to note the continued abnormal erythrocyte sedimentation rate and formol-gel reaction, despite the apparent clinical inactivity of infection over long periods. A disturbing feature of this case was the collapse after the fifth and sixth serum injections.

*Case 13.*—J. E. B. (MX. 57652. 485/39), aged 16 years.

*Family history.*—Nothing relevant to rheumatism.

*Previous history.*—Tonsillitis infrequent—no rheumatism.

*Present history.*—19.1.39: Effusions in both knee-joints, particularly the left. Elbow-joints tender and painful.

20.1.39: 10 c.c. convalescent serum intramuscularly.

21.1.39: Joint pains ceased but temperature still elevated. 10 c.c. convalescent serum intramuscularly.

24.1.39: Temperature still raised—salicylates started.

25.1.39: Temperature falling.

30.1.39: Salicylates stopped.

Remainder of convalescence uneventful. Heart apparently escaped damage, and he was discharged fit for duty.

*Comment.*—Although possibly contributing to the relief of pain, serum had no effect on temperature nor on the general condition. Only a small dosage was used.

*Case 14.*—T. A. M. (SSX 26242. 893/39), aged 17 years.

*Family history.*—Mother has had rheumatic fever. Father and two siblings alive and well.

*Previous history.*—No rheumatism—tonsillitis infrequent, but last attack within six weeks of admission.

*Present history.*—Complained of aching in all joints.

10.2.39: Admitted with both knees and ankle-joints swollen and tender. Intramuscular injection 10 c.c. convalescent serum, repeated in afternoon.

11.2.39: Pains easier but still pyrexial. Intramuscular injection of 10 c.c. convalescent serum.

13.2.39: Condition unchanged and salicylates started. Good response to salicylates.

Remainder of convalescence uneventful, with no evidence of carditis.

*Comment.*—Serum had no effect in this case, which showed a good response to salicylate therapy.

Case 15.—E. P. M. (MX. 58162. 3486/39), aged 26 years.

*Family history.*—Nothing relevant.

*Previous history.*—Nothing relevant.

*Present history.*—25.5.39: While pulling in tug-of-war felt shooting pain in groin.

26.5.39: Both ankles stiff and sore.

27.5.39: Admitted to hospital with knees and ankles swollen and very painful—salicylates started.

5.6.39: Both knees affected—salicylates restarted. Good response but abnormal erythrocyte sedimentation rate and formol-gel reaction.

20.6.39: Left knee swollen and tender—colsulanyde given.

24.6.39: Both knees again active—intramuscular injection of 20 c.c. convalescent serum—pains eased and temperature remained at same level, i.e. no afternoon rise.

25.6.39: Shoulders and arms painful. 10.10 a.m. 20 c.c. convalescent serum intramuscularly; 11.20 a.m. rigor and collapse with cyanosis, shallow respirations, and feeble pulse. Adrenaline and coramine; 11.35 a.m., greatly improved and 11.55 a.m. no further distress; 1 p.m. sweating profusely—no further trouble.

26.6.39: Slight pains in both shoulders but improved.

No further joint pains, and remainder of convalescence uneventful.

*Comment.*—This case illustrated the symptomatic relief given by salicylates without prevention of relapses. In the second relapse colsulanyde was not beneficial. The first serum injection had a slight effect, but the second dose was followed by temporary collapse. This was the second case in which such a phenomenon was encountered.

Three of the five subjects in this second group have returned to duty without carditis. One patient is still convalescent with both mitral and aortic lesions, and one patient has returned to duty with a mitral lesion.

#### DISCUSSION

This paper records the results of a preliminary investigation on the possible use of convalescent serum in the treatment of acute rheumatism. As the clinical records show, there was a tendency for the initial pyrexia to be of short duration, and this rendered difficult any estimation of the effect of serum on an easily recognized sign. Nevertheless, when given in the early stages of an attack, serum did appear to reduce the period of pyrexia, and this was particularly noticeable in primary attacks. Clinical study of the cases has left no doubt that arthritic pain was relieved in such cases. Of the 15 cases treated in all, nine were considered to have benefited. Other antisera have been used in the treatment of rheumatism. Thus antistreptococcal serum has been tried with varying success. Wilson (1930) and Hill (1928) reported adversely on its use, while Toogood (1926), Easson and Thomson (1934), and Small (1928) were of the general opinion that serum was of value.

The volume of serum in the present investigation was not large enough to permit really adequate dosage in every case, but the results obtained justify further extension of the method on the lines suggested.

One point which will require careful investigation, was the occurrence of partial collapse in two individuals within a short period of receiving an injection of serum. The cause of this peculiar phenomenon has not been discovered. Anaphylaxis was considered, but the time relations did not support this possibility. That it was not due simply to repeated dosage has been shown by the absence of any untoward reaction in patients receiving similar courses. Nor could any individual batch of serum be incriminated since two separate batches were concerned, and other patients

treated with the same sera have not been affected. Similar reactions have been reported by Hitchcock, McEwen and Swift (1930) following the use of antistreptococcal serum and by Poynton and Schlesinger (1937). In their cases the serum was obtained from heterologous species, whereas in the present series the serum was from the homologous species. Alarming as these two incidents were at the time, they do not present a serious contra-indication as the patients rapidly responded to appropriate treatment.

It is hoped that the investigation will now be extended, and that the true value of serum therapy will be ascertained by its effect on the course of the disease and, in particular, on the prevention of cardiac lesions, when given in adequate doses in the early stages of arthritis.

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## Section of Anæsthetics

President—HAROLD SINGTON, M.D.

[February 2, 1940]

### DISCUSSION ON PREMEDICATION

**Dr. Ernest Landau :** Premedication may be broadly defined as the administration of drugs during the period immediately prior to the induction of anæsthesia for purposes connected with the administration of the anæsthetic. It is usual to prescribe one drug of the atropine series to control the flow of mucus and lessen the risk of vagal inhibition. Atropine was the drug used originally for this purpose, but scopolamine is in many ways preferable.

Apart from the action provided by the atropine series of drugs, premedication serves three purposes :—

(1) It provides psychic sedation. Except in certain cases where there is a definite contra-indication, psychic sedation is advantageous both to patient and anæsthetist. The injection of omnopon  $\frac{1}{2}$  gr., scopolamine  $\frac{1}{150}$  gr. will often convert a terrifying ordeal into a comparatively trivial incident.

(2) It will enable anæsthesia to be induced and maintained with a lower concentration of the anæsthetic employed.

(3) Its most important function is to produce a degree of respiratory depression, or more accurately, to decrease the sensitivity of the respiratory centre to carbon-dioxide stimulation, for this is the increasing of respiratory depression. Yandell Henderson, who has done so much invaluable work on the physiology of respiration, describes a cycle occurring during the administration of ether to the patient premedicated with atropine only, which I think is familiar to some of us. Ether in the lighter stages of anæsthesia increases the sensitivity of the respiratory centre, but in deep anæsthesia decreases the sensitivity of the centre. The combination of fear and light ether anæsthesia increases the sensitivity of the respiratory centre to such an extent that the normal  $\text{CO}_2$  tension will stimulate it and produce overbreathing, and when a deeper stage of anæsthesia is reached and the centre depressed, the lowered  $\text{CO}_2$  tension will be insufficient to maintain respiration and apnœa will result, even though the patient is only partially anæsthetized. During this period the  $\text{CO}_2$  tension of the blood rises and the  $\text{O}_2$  tension decreases. Anoxia increases the sensitivity of the centre and breathing begins again. Unfortunately anoxia increases the sensitivity of the centre long after the anoxia of the centre has been relieved, and a second period of overbreathing occurs, followed by apnœa, increased anoxia, and perhaps then death. A similar cycle may occur under chloroform anæsthesia, and the apnœa followed by hyperpnœa and a sudden concentration of chloroform in the blood may produce, as shown by Goodman Levy, a fatal ventricular fibrillation.

Yandell Henderson has shown that morphinization, by decreasing the sensitivity of the centre, will prevent these changes in respiratory rhythm, which are often in the atropinized patient so difficult to correct and a cause of irritation to the surgeon, anxiety to the anaesthetist, and danger to the patient.

In the morphinized patient the depressed centre is less responsive to the changes produced by light anaesthesia, induction is not accompanied by gross changes in rhythm, but the anaesthesia progresses smoothly through the various stages until the requisite plane is reached, and the patient soon settles down to a smooth, quiet, and regular rhythm.

The extent to which depression of the respiratory centre is to be permitted will vary according to the method of anaesthesia, the type of operation to be performed, and the age and condition of the patient. At one end of the scale is the administration of nitrous oxide-oxygen in which a maximum of depression and lowering of the basal metabolic rate is essential if smooth anaesthesia is to be maintained without supplementary drugs. Here premedication may be so heavy as almost to warrant the description of basal narcosis. McKesson has stated that in nitrous oxide-oxygen anaesthesia half the anaesthetic is borne by the premedication. It is true that minor degrees of anoxia may occur under gas-oxygen anaesthesia with morphine premedication, owing to the decreased sensitivity of the centre to oxygen-want, and it is for this reason that it is so essential to check the blood-pressure, pulse-rate, and respiratory rate at frequent intervals during this form of anaesthesia.

At the other end of the scale is premedication before spinal anaesthesia. Sedation is desirable with a minimum of depression; here of course the premedication plays no active part in the anaesthesia. My own preference and that of most of the surgeons for whom I work is for spinal anaesthesia to be accompanied by light narcosis either with nitrous oxide-oxygen, cyclopropane or pentothal. Much of the collapse occurring during spinal anaesthesia is psychic in origin and certainly with light premedication, light narcosis and a liberal supply of oxygen, the condition of the patient seems to be better than when premedication alone is used. Premedication should be light because the effect of the drugs used for spinal anaesthesia is unpredictable; sometimes the combined effect of light premedication, the fall in blood-pressure, and the absorption of the drug used for the spinal anaesthetic into the blood-stream, is to produce a light narcosis, and the patient may sleep throughout the operation. If heavy premedication is used, the additional depression produced by the spinal anaesthetic may prove embarrassing. It is safer to premedicate lightly and, if necessary, either supplement the premedication with light narcosis, or give a supplementary injection of the drugs used for premedication.

Although on theoretical grounds morphia is a desirable safeguard if open ether is to be given, the respiratory depression produced may be such that induction and maintenance of anaesthesia become difficult and post-operative recovery prolonged. It is for this reason that the semi-closed or closed method with gas-oxygen is preferable. Anaesthesia can be induced rapidly with gas and oxygen and ether added in minimum quantities until the requisite depth of anaesthesia is reached. By careful technique and proper control of rebreathing, quiet, even anaesthesia may be obtained without subjecting the patient to saturation with ether. If chloroform *must* be used atropine and morphine are invaluable.

With cyclopropane-oxygen anaesthesia, a rather lesser degree of depression is desirable. Cyclopropane is a respiratory stimulant in light anaesthesia. The stimulus to respiration will be depressed by the lowering of  $\text{CO}_2$  tension in the closed circuit and the sensitivity of the centre lowered by the high concentration of oxygen used with this gas, therefore only a minimal degree of premedication is required. Owing to the very rapid induction the respiratory stimulating effect of cyclopropane is not very noticeable, the centre rapidly becomes depressed so that over-breathing should

not occur even with very light premedication; my standard dose for an adult is omnopon  $\frac{1}{8}$  gr., scopolamine  $\frac{1}{150}$  gr.

Premedication used before the intravenous barbiturates, whether they are used merely as basal narcotics, or complete anæsthetics, will permit smaller quantities to be given to attain the desired result. Providing that the first 4-5 c.c. are given extremely slowly with a short interval to allow the patient to recover from the preliminary extreme respiratory depression, which is a marked feature of pentothal narcosis, I have never had any anxiety concerning the patient's respiratory centre; there is, however, a longer recovery period following the preliminary injection of morphine, and if this is considered to be undesirable the premedication may be omitted. Any drugs used should be given at such a time as to ensure that their action is ended or at any rate practically finished before the patient is returned to bed. It is for this reason that I feel that such combinations as omnopon-scopolamine, followed by avertin, except perhaps in thyrotoxic patients with a high metabolic rate, or morphine-scopolamine followed by deep ether anæsthesia, are not entirely safe.

The change from atropine to atropine and morphine marked an important step in the realization by anæsthetists of the advantages of a small degree of depression. The addition of morphine helps to minimize the undesirable side actions of atropine, namely its cerebral excitant action, its effect of raising the basal metabolic rate, respiratory and pulse-rate. I use this combination for elderly patients as it produces on the whole rather less depression than omnopon-scopolamine, though the morphine may on occasion be responsible for some vomiting and nausea. *Scopolamine* differs chiefly from atropine in that it is a cerebral depressant and augments rather than neutralizes the action of morphine. It is therefore of greater value for sedation. It has a rather shorter action than atropine, and may be less liable to encourage the formation of the thick plugs of mucus which some authorities suggest are responsible for post-operative pulmonary collapse.

Before gas-oxygen anæsthesia, where maximum depression is required, I use morphia-scopolamine in what may be considered maximal doses, and before other forms of anæsthesia omnopon-scopolamine in doses ranging from  $\frac{1}{8}$  gr. to  $\frac{3}{8}$  gr., with scopolamine  $\frac{1}{150}$ . Omnopon contains half its weight of morphine plus 20% narcotine and other alkaloids of opium; it is less depressant and rather more sedative than morphine. For children between the ages of 14 and 18, and adults over 60 I give a half-dose of omnopon-scopolamine. A reduced dose will also be given to very ill or toxic patients to avoid over-depressing the centre. It is better to give a small dose and repeat it if necessary rather than over-drug the patient. These injections should be given one and a half hours before operation so that the maximum effect is exerted at the commencement of the anæsthesia and not during the later stages of the operation or after the patient's return to bed when further depression is most undesirable. Although smaller doses of these drugs may be given to younger children, the fractional doses prescribed ranging from omnopon  $\frac{1}{16}$  and scopolamine  $\frac{1}{150}$  gr. may lead to mistakes in administration, and for this reason I generally use nembital by mouth with atropine. I have tried many of the products put on the market by enthusiastic manufacturers, and in my experience nembital is the most valuable. Its action is rapid and short, and fairly, though not absolutely, reliable. The acid salt is insoluble in water, and the nembital given preferably powdered in jam or honey, should be followed by a small draught of soda bicarbonate. The dose varies from 0.5-0.7 gr. per stone body-weight, given one hour before operation. Nembital is made up with lactose and  $1\frac{1}{2}$  gr. of nembital is contained in 2.3 gr. of the powder.

Before prescribing any of these drugs the patient and his medical attendant should be asked whether he has an idiosyncrasy to any of them. If morphia is not

tolerated omnopon, heroin, or dilaudid may be; if there is any doubt, some other drug must be substituted.

Premedication should be withheld from patients undergoing operations on the brain owing to the degree of respiratory depression which may occur during the operation. Atropine only is permissible in these cases. No morphine or barbiturates should be given prior to Caesarean section. Here we are concerned with the respiratory centre of the baby. The effect of administering these depressant drugs to the mother, even in minimum doses, is to lower the sensitivity of the respiratory centre of the newborn baby to carbon dioxide, and may result in the birth of an apnoeic baby, and even though respiration may be stimulated with carbon dioxide and oxygen mixtures, should the baby survive it runs the risk of pulmonary collapse and pneumonia. In midwifery no morphine or barbiturate should be given, for the same reason, within three hours of the birth of the baby.

For operations following which an active cough reflex is essential the premedication and anaesthetic must be planned so that these reflexes are active before the patient leaves the theatre. I do not believe that adequate premedication correctly timed adds to the post-operative risk.

The depressant effect of morphine, if this needs any further justification, is more than compensated by the decreased amount of anaesthetic required, and therefore in the early return of muscular tone.

It is essential that great care be taken in the choice of the drug and its dosage, and that the chosen drugs be given at the right time.

**Dr. J. U. Human:** For pre-operative medication the principal drugs I shall consider are atropine, scopolamine or hyoscine, morphia, omnopon, and nembital by mouth and other drugs will be mentioned incidentally.

The general rule in pre-operative sedation should be to give less of the hypnotic drug the greater the operative risk. Safe procedures such as tonsillectomies and plastic operations about the face should have full basal narcosis; whereas poor-risk cases such as gastrectomies should have  $\frac{1}{100}$  gr. of atropine only.

With the apparatus now available we can administer pure nitrous oxide until consciousness is lost, and it is seldom that more than six to twelve breaths of this easily inhalable gas are required before the eyelash reflex goes. Ether can then be added with no discomfort to the patient. With poor-risk cases, therefore, one should not hesitate to sacrifice the patient's comfort in the interests of safety, for should shock occur during the operation an hypnotic dose which appeared to be perfectly safe in the early part of the operation will now become a dangerous dose. With the exception of paraldehyde the pre-operative narcotic drugs are not eliminated by respiration, but by the patient's metabolism.

The higher the metabolic rate of a patient the more sedation will be required and fat subjects need relatively less hypnotic premedication than thin subjects because obesity is generally accompanied by a low basal metabolic rate.

It should be remembered that an ill patient is already premedicated with his own toxins, and although a raised temperature means a raised metabolic rate, hypnotic premedication should be sparingly given, or entirely withheld.

*Atropine* is different from all the other premedicating drugs in that it produces no sedation and, consequently, does not reduce the amount of the inhalation anaesthetic required. It increases metabolism and therefore it makes the patient require more of the anaesthetic for a given depth of anaesthesia. This is because the basal oxygen requirements are increased.

The real value of atropine lies in its ability to reduce or to prevent an excessive secretion of mucus during an ether administration, but it is also said to have some

action on the vagus which renders the ventricles of the heart less liable to fibrillation during a chloroform induction. Atropine can, therefore, be regarded as a therapy comparable with the pre-operative administration of glucose and insulin when a diabetic has to be operated upon. The patient is simply put in a more physiologically fit condition to be anæsthetized.

The dosage of atropine is simple, and patients should be divided into two age-groups :—

From birth until 6 months =  $\frac{1}{200}$  gr.  
 From 6 months and over =  $\frac{1}{100}$  gr.

After injecting  $\frac{1}{100}$  gr. of atropine the child of 1 year is often more bubbly and moist than an adult, for children, being more "watery" in their make-up, need relatively much more atropine to dry up the secretions. These two doses should, of course, be varied to suit individual cases. For example, a big fat patient aged 40 with chronic bronchitis should have  $\frac{1}{8}$  gr. or even as much as  $\frac{1}{50}$  gr., whereas a desiccated old patient of 80 with normal lungs should have the dose reduced to  $\frac{1}{150}$  gr.

Atropine is useful for teaching purposes, for the pupils dilate readily as anæsthesia descends and the respiratory signs of anæsthesia are also accentuated. When demonstrating the signs of anæsthesia the resistant atropinized patient will descend slowly enough for the anæsthetist to be able to demonstrate all the signs of anæsthesia.

Atropine should never be given before Cæsarean section; if no premedication of any kind is given, most of these operations can be performed under  $N_2O + O_2$  only. Muscular relaxation is not required here, and even when the peritoneal edges are sewn together there will be so much "slack" in the walls of the now relatively empty abdomen that no difficulty will be encountered by the surgeon. Atropine raises the metabolic rate of the patient so much that it is usually impossible to maintain first-plane anæsthesia with  $N_2O + O_2$  alone, but without atropine, even if a trace of ether vapour is required for the first few minutes, maintenance on  $N_2O + O_2$  only will be easy. Most of the mucus is secreted from above the level of the vocal cords and with a very slight Trendelenburg position the mucus will gravitate away from the trachea and bronchi and can easily be evacuated from the mouth by turning the head on one side and lifting the lower edge of the mask on expiration when required.

With the exception of paraldehyde, no hypnotic premedication of any kind should be given before childbirth because a newly born infant is very reluctant to breathe when the mother has had one of these drugs, particularly morphia or omnopon.

Two points to remember :—

- (a) Never give less than  $\frac{1}{100}$  gr. of atropine to a normal child over 6 months of age.
- (b) Never give atropine when an attempt is to be made to give  $N_2O + O_2$  only.

*Morphia and omnopon* by themselves have very little hypnotic effect, but they relieve pain and lower metabolism and therefore less of the subsequent inhalation anæsthetic will be required. By themselves they have a very slight drying-up effect, but they are powerful respiratory depressants. Not more than  $\frac{1}{150}$  gr. of atropine need be combined with  $\frac{1}{4}$  gr. of morphia.

Morphia and omnopon are, however, invaluable when a good-risk operation is to be performed on a healthy adult under  $N_2O + O_2$  only. If diathermy is to be used about the head or neck, endotracheal gas and oxygen can be used and both ether and chloroform can be avoided.

When the anæsthetist is confronted with a very resistant dental patient an intravenous pupil-contracting dose of morphia or omnopon is a very great help. If it is known from previous experience that the patient is difficult, omnopon can be injected into the median basilic vein, and I have found the pupils a sure guide to the

dosage. A very small dose will sometimes be sufficient. Charge the syringe with  $\frac{2}{3}$  gr. of omnopon and inject very slowly into the vein while watching the eyes, and as soon as the pupils contract withdraw the needle. The resistant patient will now be transformed into an easy subject for nasal gas.

After a pupil-contracting dose of morphia or omnopon only, the patient will be able to go home alone in a cab.

*Hyoscine or scopolamine* has two big advantages over atropine :—

(a) It dries up the secretions much more effectively than does atropine.

(b) It is also a hypnotic and by reducing metabolism less of the subsequent inhalation anæsthetic is required.

But hyoscine is even more limited in its use than morphia and omnopon, for not only must the patient be healthy and the operation a good risk, but he must be neither very young nor very old. I once gave  $\frac{1}{3}$  gr. of omnopon plus  $\frac{1}{150}$  gr. of scopolamine to an old woman before giving gas and oxygen for a dental operation and she remained unconscious for two days.

If I cannot see the patient pre-operatively and hyoscine is suggested, I restrict its use to between the age-limits of 16 and 65. Hyoscine is rather apt to cause restlessness and excitement, pre-operatively as well as post-operatively. But this restlessness can be controlled by combining it with an adequate dose of another sedative drug such as morphia, omnopon, or nembutal. I have never found restlessness to occur when omnopon and scopolamine are combined in the proportions of  $\frac{2}{3}$  gr. of omnopon to  $\frac{1}{150}$  gr. of scopolamine, but with only  $\frac{1}{3}$  gr. of omnopon plus  $\frac{1}{150}$  gr. of scopolamine I have seen restlessness.

The hypnotic effect of  $\frac{2}{3}$  gr. of omnopon plus  $\frac{1}{150}$  gr. of scopolamine given one hour before the operation is very good, and can be injected by the nursing staff. Post-operative vomiting, however, is very often troublesome after morphia or omnopon, but this can be reduced to some extent by giving a few cubic centimetres of pentothal sodium intravenously just before the operation, as this drug seems to contain some anti-vomiting principle.

As  $\frac{2}{3}$  gr. of omnopon plus  $\frac{1}{150}$  gr. of scopolamine lowers metabolism more than a basal narcotic dose of avertin, toxic thyroids do very well after this premedication. But the injection should be given only three-quarters of an hour before the operation as these patients very rapidly break up and eliminate any drugs. Combined with some local anæsthesia thyroids can be done in this manner under  $N_2O + O_2$  only.

*Nembutal*, in capsule form by mouth, can be given by the nursing staff in the absence of the anæsthetist.

Nembutal is more slowly metabolized and eliminated than evipan or pentothal and therefore has a rather more prolonged hypnotic effect.

The usual dose for a healthy adult is 3 gr. by mouth one hour before the operation, and if the operation is in the morning  $1\frac{1}{2}$  gr. can be given the previous evening as well. A total dose of  $4\frac{1}{2}$  gr. should, however, never be exceeded. After 3 gr. of nembutal the patient will often be presented to the anæsthetist in a very wideawake condition and he will sometimes spontaneously remark that the "dope" has had no effect whatsoever upon him, yet when the inhalation anæsthetic is administered remarkably few breaths of  $N_2O$  will be required to produce unconsciousness. After three or four breaths the eyelash reflex generally goes. It is interesting to compare this with omnopon and scopolamine, for where  $\frac{2}{3}$  gr. of omnopon plus  $\frac{1}{150}$  gr. of scopolamine has been injected one hour before operation, though the patient enters the theatre in a very drowsy condition and answers questions only after they have been repeated, many more breaths of  $N_2O$  will be required before consciousness is lost. Subsequently, less anæsthetic is required whichever drug has been used for premedication.

Nembutal is sometimes quite ineffective an hour afterwards because at this time patients are generally nervous, and nervousness inhibits digestion and absorption from the gastro-intestinal tract. When, however, an injection of  $\frac{1}{150}$  gr. of scopolamine is given at the same time as 3 gr. of nembutal is given by mouth there is always a very profound hypnotic effect an hour later, for the injected drug allays nervousness and gastric inhibition is thus removed. The patient will be considerably drier than when  $\frac{1}{100}$  gr. of atropine is injected with the nembutal.

Good-risk cases do exceedingly well when nembutal by mouth has been the only pre-operative sedation, and the drug, *per se*, does not seem to cause any post-operative sickness.

Children often have much post-operative restlessness after nembutal, and one not infrequently encounters the aggravating type of case where, after the full dose by mouth, there is no visible sedation an hour later; but after the operation a delayed action, or absorption, makes unconsciousness last for so many hours that the parents get worried.

A good dosage rule for normal children is to give  $\frac{1}{8}$  gr. for every year of life and to modify this according to circumstances. In children 8 years of age  $1\frac{1}{2}$  gr. of nembutal by mouth one hour before will often produce a quietly sleeping child on the operating table.

It is particularly unwise to give nembutal before a urological operation, for the longer acting barbiturates have a tendency to cause a suppression of urine in these patients.

In my experience nembutal is the ideal premedicating agent when cyclopropane is to be given for a good-risk operation.

Never synergize with the barbiturates, for an unexpectedly profound, or even fatal, narcosis might result. The anæsthetist should leave precise instructions for medication with the nursing staff in case the patient cannot get off to sleep on the night before the operation. It is unwise, for example, to help a patient to sleep with 5 gr. of medinal when it is known that nembutal, evipan, or pentothal is to be given in the morning. It is quite safe to combine a barbiturate with other hypnotic drugs, but the rule of one barbiturate at a time should be rigidly adhered to.

**Dr. F. Barnett Mallinson:** Premedication in anæsthesia gives the following advantages:—

*To the patient.*—(1) Reduction of apprehension and increase of pre-operative comfort. (2) Lessening of after-pain. (3) Decreased incidence of post-operative vomiting.

*To the surgeon.*—(1) More easily obtained muscular relaxation. (2) Quieter respiration during anæsthesia. (3) Considerable reduction of psychic shock, and in children especially, of the effects of psychic trauma.

*To the anæsthetist.*—(1) A smoother and quicker induction is obtainable. (2) The increased mucous and salivary secretions which result from ether and ethyl chloride are counteracted. (3) Less of the anæsthetic drugs are needed; an especial advantage if using toxic agents such as ether and chloroform.

Outstanding disadvantages are: (1) With some techniques there is added labour for all concerned; but this is surely worth while in view of the benefits to be gained. (2) The depressed respiration which results has been accused of increasing the likelihood of post-operative pulmonary complications, but the risk is slight, especially since the introduction of carbon dioxide to the anæsthetist's armamentarium. (3) There is frequently some degree of sluggishness of reflexes post-operatively. Care in choice of technique can largely eliminate this when brisk reflexes are desired after operation. (4) The degree of depression of the heavily premedicated patient's

respiratory system after return to the ward may constitute a danger. Here again we now have carbon dioxide and oxygen and an experienced nursing staff. When, however, we have large numbers of seriously shocked and narcotized patients resulting from air raids, great caution will need to be exercised as the nursing staff will be hard pressed.

The administration of glucose before operation should never be omitted. It is invaluable for buttressing the liver against the assault of the more toxic agents and thus diminishing post-operative "acidosis."

Atropine, the use of which as a premedicant dates from the early years of this century, is of the greatest importance, and should always be given except when hyoscine be used. Atropine should then be omitted, because not only will hyoscine do the work of atropine but, as has been pointed out by many observers including Nosworthy and Guedel, the narcotic action of morphine and hyoscine is to a considerable extent counteracted by atropine.

Advantages accruing from atropine are: (1) It dries up the mucous and salivary secretions stimulated by ether and ethyl chloride. (2) It acts on the vagus nerve and is thus said to diminish the likelihood of vagal inhibition of the heart. (3) It diminishes the secretion of adrenaline, excess of which is widely held responsible for ventricular fibrillation during the fear stage of induction.

The chief disadvantage of atropine is its stimulating effect on metabolism, resulting in what Guedel terms a "higher starting point" for the induction of anaesthesia.

The dose of atropine employed is important. Adults frequently get too big a dose, while children, who tolerate the drug particularly well and in whom ether is used much more, often get too little. An adult dose of  $\frac{1}{50}$  gr., as frequently prescribed, makes one regard atropine as a possible contributor to that form of heat-stroke which some observers, notably Dickson Wright, and Woolmer and Taylor, consider to be the cause of so-called "ether convulsions". A maximum of  $\frac{1}{100}$  gr. is therefore preferable and adequate. Children, however, get the adult dose down to 1 year of age;  $\frac{1}{150}$  gr. down to 6 months; and  $\frac{1}{200}$  gr. below that.

Atropine combined with morphine  $\frac{1}{8}$ – $\frac{1}{4}$  gr. has been much used and produces to a certain degree all the advantages enumerated above except that in a certain number of cases vomiting is increased. Therefore omnopon is substituted for morphine and is found to effect an improvement. Furthermore hyoscine, which will do all the work of atropine, has the additional advantages of enhancing the effect of omnopon and of having a less stimulating effect on metabolism. Omnopon  $\frac{2}{3}$  gr. and scopolamine  $\frac{1}{150}$  gr. give much better results and can be employed safely for all fit patients between the ages of 18 and 65 years. For older, enfeebled, or shocked patients,  $\frac{1}{8}$  gr. and  $\frac{1}{150}$  gr. are perhaps wiser. The La Roche preparation of both drugs together is particularly effective.

The combination of omnopon and scopolamine, however, should not produce complete unconsciousness by itself. While this is convenient for hospital routine and long lists, there is no doubt that full unconsciousness is often preferable and is necessary to get the maximum benefit from pre-anaesthetic medication in the directions of comfort for the patient and reduction of psychic shock. This brings up the problem of basal narcosis, in many ways the pre-anaesthetic ideal. In the light of this problem premedication has to be modified.

*Basal narcosis* can be accomplished by three routes: (1) By mouth; (2) *per rectum*; (3) intravenously.

*By mouth.*—Nembutal is the drug most commonly used to produce basal narcosis in this way. It is usually given in doses of 3–4½ gr. and atropine should also be given for the reasons mentioned before. Alone nevertheless, in adults at any rate, nembutal

is apt to be exceedingly unreliable. It is more certain when combined with omnopon and scopolamine, but is then as a rule far too depressant for comfort or safety. Nembutal seems to be much more satisfactory, however, in children, a safe and fairly effective technique being  $\frac{1}{2}$  gr. per stone of body-weight up to 3 gr. It is useful for long hospital lists, requiring the minimum of preparation and technique.

*Rectal administration.*—Nembutal may also be given *per rectum* in children. A dosage of  $\frac{3}{4}$  gr. per year of age up to 8 years seems safe and perhaps more certain than oral administration.

Paraldehyde is more reliable and may be given in doses of 1 drachm per stone of body-weight. In adults it should be combined with omnopon and scopolamine to get the best results, and given two hours before operation owing to its slowness in action. It is very safe and used alone, except for atropine, as in children, has almost no depressant effects. Its main disadvantages are a rather prolonged action and its distinctive odour.

The best drug for rectal administration is without any doubt avertin. It is absolutely certain in action and has a high degree of safety if used with discretion in selection of cases and in dosage, being particularly well tolerated by children. A dose of 0.09 grm. per kilo for females and 0.1 grm. per kilo for males and in children, given half an hour before operation, is both safe and reliable. Omnopon and scopolamine are sometimes combined with avertin, but as a rule this practice can only reduce its excellent margin of safety and should never be made routine. When brisk reflexes are essential after operations such as tonsillectomy, paraldehyde may be preferable. The chief disadvantages of avertin are the somewhat exacting technique of preparation and testing which is required, and the necessity for rectal interference.

*Intravenous injection.*—One of the safest and perhaps the most effective method of producing basal narcosis is the intravenous injection of a barbiturate. This is best combined with a preliminary injection of omnopon and scopolamine  $\frac{1}{3}$  gr. and  $\frac{1}{150}$  gr. and results in a smoother and quicker induction with less barbiturate. Furthermore, its effects last longer. Omnopon  $\frac{3}{4}$  gr. and scopolamine  $\frac{1}{150}$  gr. is apt to be too depressant.

The ease and simplicity of administration and the astonishing absence of real contra-indications have led one to adopt the intravenous route as a standard technique other factors being favourable. The two contra-indications which do seem to be of importance are:—

(1) Asthma. I have seen a patient nearly die of acute status asthmaticus after 1 grm. of evipan.

(2) Patients in whom the liver may be so damaged as to be incapable of detoxicating the drug properly, such as in prolonged serious sepsis.

Of the two quick-acting barbiturates which show a real advance in efficiency, pentothal has the following advantages over evipan: (1) Smoother, quicker induction; (2) freedom from laryngeal spasm; (3) jactitation never occurs; (4) more powerful and reliable action.

Nembutal intravenously has the following serious disadvantages which are intensified if omnopon and scopolamine are added: (1) Prolonged action; (2) post-operative restlessness; (3) poor controllability owing to slow detoxication.

With pentothal the dose of omnopon and scopolamine should be given one and a half hours before the barbiturate. This gives the depressant effect of the hyoscine time to wear off somewhat, while not vitiating its amnesic value. Patients who have received this premedication rarely resent and frequently do not even notice the subsequent venupuncture.

## SPECIAL CONSIDERATIONS

It is suggested that the maximum benefits are obtained from pre-anæsthetic medication if complete unconsciousness is attained. There are cases in which basal narcosis may well be said to be absolutely essential.

Outstanding amongst these are :—

(1) *Children*.—No child should ever go to the anæsthetic room while conscious. It is strongly felt by many that the psychic trauma may be irreparable and manifests itself almost invariably if another anæsthetic is required during childhood and frequently persists throughout life.

(2) *Thyrotoxicosis*.—Severe cases of hyperthyroidism are immeasurably more difficult to manage and less likely to survive operation without full basal narcosis. Avertin for these cases is the method of choice and has the advantage, in theory at any rate, of being antagonistic to thyroxin. The dose requires to be high, 0.11–0.12 grm. per kilo, and can often be combined with omnopon  $\frac{1}{2}$  gr. which helps to reduce the basal metabolic rate and smoothen anæsthesia.

(3) *Spinals*.—To submit the apprehensive pre-operative patient to a spinal tap followed by such manipulations as attend the administration of spinal analgesia while he is conscious must contribute a good deal to the production of psychic shock. Premedication with omnopon and scopolamine followed by pentothal makes the job quicker and easier and no local anæsthetic is needed. Jarman points out two further advantages which can be claimed for this method and which have been borne out by considerable personal experience : (a) The lessened risk of respiratory disturbance, retching, &c. ; (b) the diminished incidence of post-operative headache. All that remains to be done afterwards is to add a light gas-and-oxygen anæsthesia, which aided by the pentothal allows a high percentage of oxygen to be inhaled. This is of material assistance to a patient whose respiration is handicapped by a high spinal anæsthetic, possibly with the Trendelenburg position.

(4) *Cranial surgery*.—The use of premedication other than by atropine in cranial surgery, especially in operations associated with posterior fossa lesions, requires great caution. No heavy premedication is permissible, as being too dangerous to a respiratory centre whose function may already be seriously threatened. A small dose of omnopon not exceeding  $\frac{1}{2}$  gr. may be used with caution in selected patients, but hyoscine is considered too dangerous. Avertin is favoured by some surgeons and lessens hæmorrhage by causing a moderate reduction in blood-pressure. Others, however, fear its respiratory depressant action more.

## SUMMARY

- (1) The benefits of glucose should never be forgotten.
- (2) Atropine is of the greatest importance in premedication except when hyoscine is used, when it must be omitted.
- (3) Omnopon and scopolamine form the most satisfactory combination for hypodermic premedication.
- (4) An intravenous barbiturate to follow (3) is suggested as the ideal standard technique.
- (5) For children, who should never leave their beds conscious, avertin is excellent and very safe. Paraldehyde is a useful alternative where active reflexes are required after operation. Nembutal by mouth or *per rectum* is fairly reliable during long lists when time is short.
- (6) Amongst other special cases where basal narcosis is essential are thyrotoxic patients who do very well with avertin.

(7) Lastly, an example of the type of work in which, owing to the special dangers present, premedication must be greatly modified, is cranial surgery.

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**Dr. F. F. Waddy** said he did not agree that premedication was essential in all children. While agreeing that it was necessary for a few children, particularly those who had been frightened previously by a clumsily administered anæsthetic, he maintained that the vast majority of children could be anæsthetized without protest if approached properly. His own method was to use an open mask and two bottles of ethyl chloride scented with lavender and "eau de Cologne". The secret of the method was to tell the child what was going to happen, to let him think that he was getting his own way by choosing, and to let him hold the mask if he desired to do so. The anæsthetist should stand in front of the child and no bottle or other object should be touched by him or the nurse except in full view of the child. Everything used should be shown to him first.

As to atropine, on four occasions patients to whom he had intended to administer a spinal anæsthetic, because of chronic bronchitis, had recovered without any sign of respiratory discomfort from ether, administered without preliminary atropine on failure to perform the spinal puncture. This had led him recently to administer ether to old patients with chronic bronchitis without any preliminary medication with gratifying results. These patients suffered unless they were able to expectorate, and drying up their secretions left a viscid troublesome mucus.

Speaking of nembutal he reminded the Section of Dr. Featherstone's suggestion (*Proc. Roy. Soc. Med.*, 1934, **27**, 109) that the failure of nembutal to act might be due to spasm of the pylorus in nervous subjects and suggested that this was the true reason for giving  $1\frac{1}{2}$  gr. of nembutal the night before operation in addition to the appropriate pre-operative dose. The speaker recommended in suitable cases the further splitting of the dose of nembutal, suggesting that for late morning or afternoon operations  $1\frac{1}{2}$  gr. at night,  $1\frac{1}{2}$  gr. at breakfast in the morning (tea and toast), together with 3 gr. an hour before operation was a very effective method of dosage.

He agreed with many other anæsthetists that the larger doses were usually responsible for post-operative restlessness. He suggested the post-operative administration of glucose and atropine to combat this effect, quoting a case in which a patient who was quite uncontrollable recovered within half an hour of the administration of glucose *per rectum* and  $\frac{3}{16}$  gr. of atropine sulphate hypodermically.

**Dr. Gerald Slot**: Death may occur after premedication either with barbiturates or a morphia-scopolamine premedication. In cases where I have performed an autopsy, no definite pathological changes have been found. There were no hæmorrhages in the brain, but there was a congestion of the bases of the lungs. How far this may have been due to the invariable attempts at artificial respiration over a long period, it is impossible to say. Without the clinical history, it would have been very difficult to determine the cause of death.

Clinically, I have no doubt that respiratory failure is the fatal factor. It may be assisted by a failure of respiration associated with the low blood-pressure which occurs in these cases. The immediate treatment would seem to be injections of picrotoxin. Tatum has shown that barbitone protects rabbits against picrotoxin and that 1 mgm.

of picrotoxin is an antidote for 30–40 mgm. of pentobarbital. Picrotoxin should be injected intravenously and continuously with ten-minute intervals. 2–5 c.c. of a 0.3% solution is used. Dose depends on the weight of the patient. American workers have found that up to 10 mgm. of the drug can be safely given. The immediate result in favourable cases is a deepening of respiration, but if the drug is given too rapidly muscular twitching may result. There is certainly a place for picrotoxin on the anæsthetic table.

It is worth while using an artificial respirator if it is easily available, and drip transfusion of 5% glucose saline may raise the blood-pressure and help kidney excretion. The rate should not exceed 20 drops a minute to avoid pulmonary congestion.

I have not found that asthmatics are specially sensitive to barbiturates. One must in asthmatics tread warily with all drugs—but in an asthma clinic under my care I frequently use phenobarbitone as a hypnotic and I have not noticed any undue sensitiveness. In avoiding lung complications post-operative care and positioning are most important.

**Dr. R. J. Minnitt** asked the openers of the discussion whether they had records of a series of cases where no premedication had been given before anæsthesia. He said that it was his practice, when the operation was short and simple, either to omit premedication altogether, or to order omnopon only, three-quarters of an hour before the anæsthetic. He found that this helped to dry up any excess secretion. He had carried out an investigation into a series of major operations in which no premedication whatever was given, and he came to the conclusion that the after-condition of the patients was better than in those cases which had been premedicated.

## Section of Urology

PRESIDENT—JOHN EVERIDGE, O.B.E., F.R.C.S.

[January 25, 1940]

### Nephro-Ureterectomy

#### PRESIDENT'S ADDRESS

By JOHN EVERIDGE, O.B.E., F.R.C.S.

Of all the structures in the body it is difficult to think of one more useless than the ureter after nephrectomy. The purpose of this address is to discuss the problems connected with its removal.

There have been few, if any, discussions upon this subject in this country since the universal adoption of excretion urography.

#### INDICATIONS FOR NEPHRO-URETERECTOMY

In a fairly wide examination of the literature upon the subject of ureterectomy one finds that practically every paper seeks either to advocate the wider adoption of the operation or to prove it is unnecessary. The greatest divergence of opinion is found around tuberculosis, the least around tumours. The convincing contribution of J. Swift Joly to the Vth Congress of the International Society of Urology answered the latter question for all time. The following indications, therefore, may be included :—

I. Epithelial tumours of the kidney and ureter.

II. Tuberculosis.

III. Obstructions at the lower end of the ureter, where hydro- or pyo-ureter is the outcome of (a) impacted stones, (b) strictures in the ureter or its meatus, (c) the obstructive pressure of vesical origin, the result of cicatrices, neoplasms, diverticula, and the systolic bladder.

IV. Congenital defects, such as duplicated ureters or ectopic ureters, especially if obstructed by stones or other causes.

V. Laxity of the uretero-vesical sphincter mechanism responsible for reflux ureteromegaly, such as may be (a) from neuromuscular dysfunction (so-called idiopathic ureteromegaly); (b) traumatic, i.e. post-operative, or following the passage of a large stone; or (c) inflammatory, usually tuberculous.

#### I. *Epithelial Tumours of the Renal Pelvis and Ureter*

Joly classified those of the kidney into benign papillomata, papillary carcinomata, transitional and squamous-celled carcinomata. Of these the papillary tumours are the most likely to produce implantation growths in the ureter or bladder, and hence to require complete ureterectomy. The same classification is adopted in the case of the ureter. Primary nephro-ureterectomy is to be carried out when practicable. It is unusual to meet patients on whom it would be dangerous to perform the complete operation; where this is so the primary two-stage operation is carried out; one should not wait until secondary transplants develop. Joly considers it preferable to remove a button of the bladder wall surrounding the intramural ureter.

*Results.*—Of 120 cases of *benign papillomata* of the renal pelvis nephrectomy was done in 85, with 4 deaths, and nephro-ureterectomy in 8, with no mortality. Of the 50 available for analysis there was recurrence in 23, 18 being in the ureter and 11 in the bladder. Secondary ureterectomy was performed in 15 cases, of which 3 remained well while 4 had further recurrences in the bladder. In only one case was there a recurrence where primary nephro-ureterectomy was performed. Removal of the lower end of the ureter appears a most important factor in diminishing risk of recurrence, and the value of the complete operation cannot be too strongly emphasized. Of 217 cases of primary carcinoma of the renal pelvis studied, the results of nephro-ureterectomy were better than of nephrectomy, in the proportion of 33% as compared to 66% recurrences, an interesting and important observation. The *situation* of ureteric growths should be noted as it supports the appeal for total ureterectomy, the figures being, upper third 12 cases, middle third 6 cases, lower third 24 cases. In 18 the tumour projected through the vesical orifice.

The predilection of tumours, whether primary or by transplantation, for the *lower end* is universally recognized. Colston, agreeing this, has likewise advocated the removal of the intramural section or, alternatively, passing a fulgurating electrode down the lumen to coagulate any deposits which may be present.

A peri-ureteral vesical deposit is preferably removed in continuity with the ureter.

Here are three cases where I have performed nephro-ureterectomy:—

*Case 1.*—Female, aged 62 (fig. 1). Primary papillomatosis in the lower third of the ureter. A peri-ureteral vesical papilloma hiding the ureter was destroyed by fulguration prior to the radical operation.

Absence of an excretion urogram and an easily palpable painful kidney indicated the need for exploration of the kidney, which was hydronephrotic, but devoid of growth.

This patient died three years later from carcinoma of the liver. She had no bladder recurrence.

*Case 2.*—Male, aged 30 (fig. 2). Primary renal papillomatosis, with a ureter packed with growth in its whole length and a transplant an inch in diameter covering the ureter orifice. I performed a two-stage primary nephro-uretero-cystectomy in January 1929.

The patient, who is now 41 years of age, has recently developed vesical papillomata which are fortunately within the scope of cystoscopic fulguration.

*Case 3.*—Male, aged 40, had been treated by me at King's College Hospital for bladder papillomata for two years by partial cystectomy followed by cystoscopic fulguration, when he developed an acute pain in the kidney. Nephrectomy was performed at another hospital, only 3 in. of the ureter being taken. A year ago a carcinoma developed in the bladder near the orifice of the ureter stump. I performed uretero-cystectomy, which has so far been successful as to the carcinoma, but he still grows papillomata.

Before studying the remaining indications we must consider how the ureter reacts to disease while still in continuity or as a functionless stump.

*Changes in the ureter after nephrectomy.*—Fagerstrom, summarizing the results of the experimental and clinical investigations of Lorin, Latchem, Otto and others, reports that the following principles seem to be established in the transformation of the ureter after nephrectomy: (1) The lumen of the normal ureter does not become obliterated by atrophy of its mucous membrane, but there is a noticeable atrophy of the muscle layers. (2) The same holds true in cases of hydro- and pyoureter, providing there is adequate drainage. (3) With obstruction the muscle layers undergo hypertrophy. (4) Ureteral peristalsis may persist for years after nephrectomy. (5) The tuberculous ureter tends to become obliterated after nephrectomy, in contradistinction to ureters infected with non-specific bacteria, which tend to remain patent. During the first two years after nephrectomy permeability of the tuberculous ureter is the rule; thereafter obliteration is to be expected. (6) Absorption of the contents of a distended ureter is very limited if it occurs at all. (7) Infection of the ureteral contents may spread through the walls and cause peri-ureteral infection and abscess formation.



FIG. 1.—Primary papilloma of the middle third of the ureter with transplants at the vesical end. Removed by juxta-vesical nephro-ureterectomy from a woman aged 62.

JOHN EVERIDGE : *Nephro-Ureterectomy.*

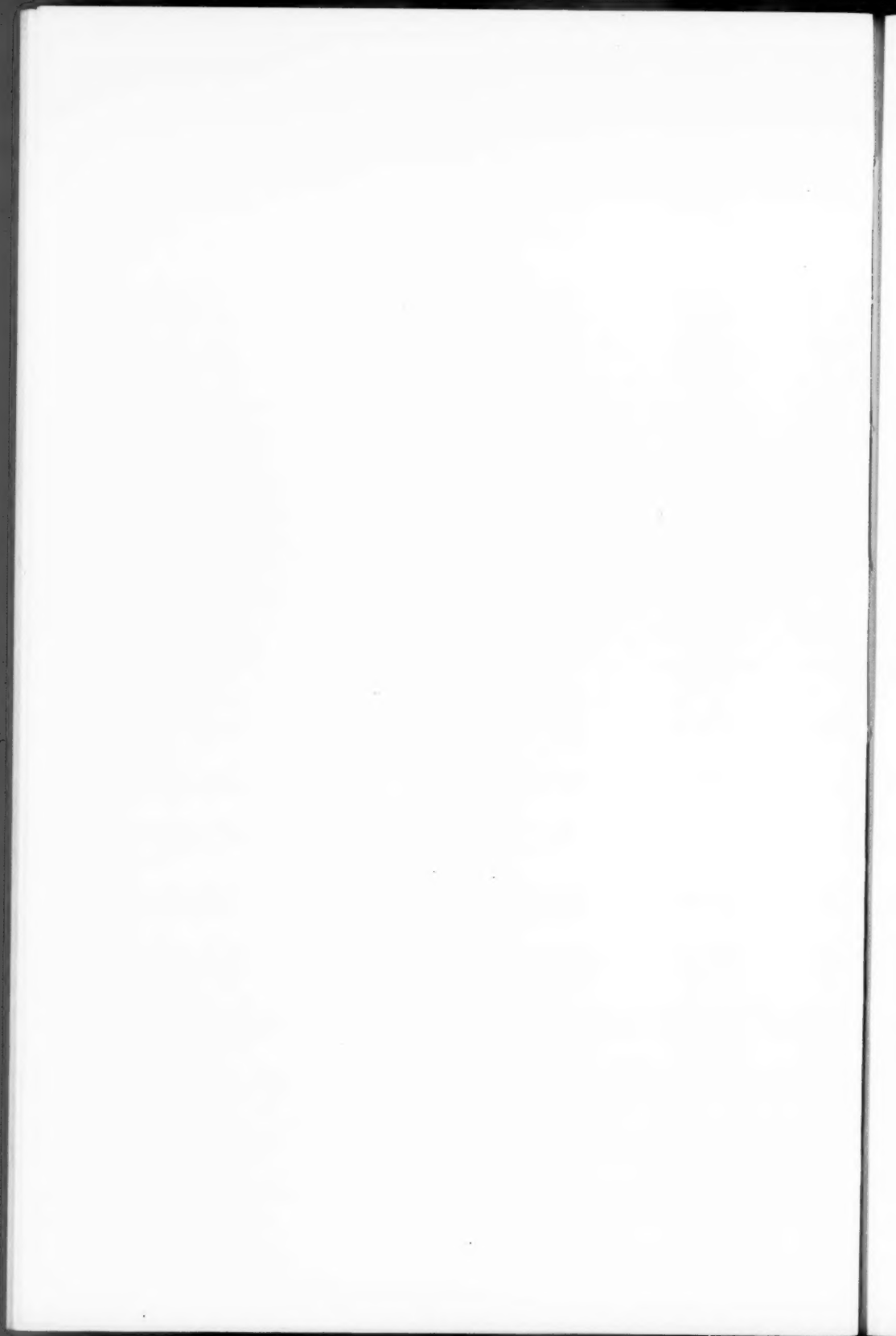




FIG. 2.—Diffuse papillomatosis of the kidney, ureter and bladder which had caused extreme hydronephrosis in a male, aged 30. Primary nephro-uretero-cystectomy was performed as a two-stage operation by the author eleven years ago. Bladder recurrence has only recently appeared.

## II. *The Ureter in Tuberculosis*

Lett in his Bradshaw Lecture described the changes in a ureter *during* active tuberculosis. An early case shows little evidence beyond thickening and vascular congestion, but if it is laid open tubercles, ulceration, or granulomatous tissue may be seen. Later it becomes much thickened, rigid, and rod-like, its length is diminished and it becomes unduly straight; cystoscopically the orifices are circular and drawn up, and urography will often show a regular permanent lumen. Such ureters are palpable sometimes abdominally, usually on pelvic examination. Great dilatation is seen in a third variety and is due either to toxic atony or to obstruction from sphincter spasm or oedema; in the later stages it may arise from a stricture in the last two inches which may be demonstrated by X-rays or obstruction to the passage of a catheter. I have found these dilated ureters to be relatively more common in

children. Narrowing may also be caused by the extension of infection from a seminal vesicle or vas deferens. Lett also mentions an atrophic variety where the ureter becomes a thin cord with a very narrow lumen, which may be partially obliterated.

My own observations from examination of a number of ureters at primary nephro-ureterectomy have shown that changes tend to be more marked at the lower end,

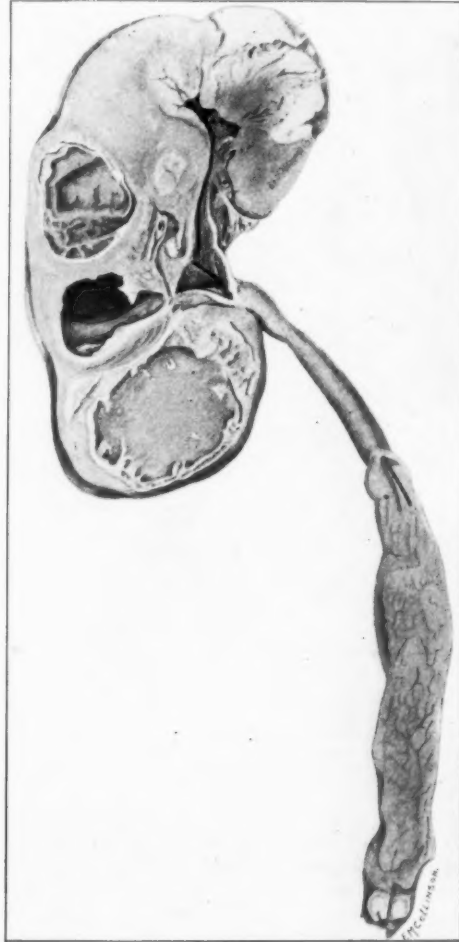


FIG. 3.—Advanced renal and ureteric tuberculosis with a breaking-down caseous focus at the vesical extremity (boy aged 17). Juxta-vesical nephro-ureterectomy performed by the author.

the mucous coat is more thickened, tubercles are distributed more copiously; one case showed a large caseous focus in the wall in the lowest inch (*see* fig. 3), and the majority exhibit a marked and massive thickening of the outer coats and a more or less severe degree of peri-ureteritis, causing adhesion to the peritoneum and to

the neighbouring vessels (the uterine and vesical arteries particularly). Several of my cases showed quite an innocent-looking ureter to the pelvic brim, and it is easy to see how one could be misled if basing an opinion upon the restricted length of the ureter coming within the scope of the lumbar field.

*Changes in the ureter stump after nephrectomy for tuberculosis.*—Those referred to by Fagerstrom I have already mentioned, they are catalogued in the majority of papers; others, however, may be added. In the early post-operative stages infection may spread to the surrounding structures, causing cellulitis and abscess formation. Disintegration of the walls may be responsible for slipping of the ligature, with resulting wound infection or a urinary fistula, especially in cases where the sphincter mechanism at the lower end has been already deranged by tuberculous inflammation allowing urinary reflux.

In the great majority of stumps the inflammatory process subsides after a variable lapse of time but in two, where I had need to perform late ureterectomy four and two and a half years after nephrectomies by other surgeons, the, approximately, 8 in. of duct I removed in either case revealed microscopically active tuberculous processes, the lumen of the former was patent in its whole length, and that of the latter in all but its highest 2 in., from which caseous debris could be expressed. Dossot refers to a ureter stump examined at autopsy by Verriotis ten years after nephrectomy; the ureter was small, the muscular tunics were atrophied, but the lumen was free and covered by epithelium. Lorin's estimation of the permeability or otherwise of the lumen was based upon the ability to catheterize at subsequent cystoscopy. In four so examined the ureter was only permeable in one, whereas after nephrectomy for calculi and hydronephrosis the lumen persisted.

Dossot has found the following complications to result from the tuberculous ureter stump: (1) Persistent cystitis. (2) Pains in the course of the ureter, which are usually recovered from in a few months. (3) Persistence of bacilluria. (4) A urinary fistula due to reflux. He refers to 10 such cases reported by Legueu and Papin. These fistulae are more common when the bladder is contracted and the ureter dilated. (Marion has obtained healing in these with the aid of a tied-in urethral catheter.) (5) A persistent purulent sinus. The last, however, may arise, according to Legueu, Marion, Thomson-Walker and Lett, from infection in the perinephric fat existent before nephrectomy.

Lett mentions further complications, and especially calls attention to the added risk of extension to the opposite kidney aggravated by the stump, in virtue of the persistent cystitis, whence ascending infection is promoted by bacilli travelling by the patent lumen of the opposite intramural ureter or by lymphatics. The opposite ureter may eventually become obstructed by a stricture or caseous deposits, and so death may ensue from renal failure, as in one of my own cases. Prolonged bladder systole may cause mechanical obstruction to the opposite ureter with hydronephrosis, as Wade and others have referred to. Lett cites three cases where he regretted leaving a ureter stump. In the first the wound broke down three weeks after operation and sinuses persisted until the stump was removed eighteen months later, when immediate healing took place. The second case had presented a practically normal bladder at the time of nephrectomy. Three months later ulceration was marked, particularly around the orifice of the stump. This persisted until ureterectomy fifteen months later, when it rapidly healed. In a third case severe colicky pains persisted up to the eighteenth month after nephrectomy, although the ureter at the time of nephrectomy appeared healthy.

It is clear from the variety of methods adopted in the past to eliminate the consequences of a retained tuberculous ureter stump that the surgeons employing them have been driven to these measures by their past experiences, thus some have attached the open ureter to the loin incision, e.g. Marion; Dor exteriorized it by ensheathing it in a gutta-percha tube. Others inject iodoform (Wildbolz), tincture of iodine (Kocher), sublimate (Kapsammer), carbolic acid (Israel). Mayo and Young

injected carbolic acid into the closed ureter stump. Legueu abandoned all such methods and relied upon division with thermo-cautery and ligature. The observation of Hunt is therefore all the more remarkable, that in the series of 574 cases where nephrectomy for tuberculosis was carried out at the Mayo Clinic in the years 1919-28, he did not find subsequent ureterectomy recorded. Occasionally the upper end of the ureter opened with contamination of the wound and subsequent indolent healing, but this was regarded as due to a technical error in the application of the ligature, and no such cases occurred in later years. Nor had any cases been observed where it seemed sufficiently certain that the persistence of cystitis and pyuria was due to persistence of infection from the ureter, and so justified ureterectomy. The only ureterectomy required in association with an infective condition of the kidney was in a single instance after non-tuberculous pyonephrosis due to a strictured ureter. Beer, on the other hand, was alive to the risk of the retained tuberculous ureter stump, and found that in 7% of his cases it was great enough to warrant primary ureterectomy. He actually performed primary nephro-ureterectomy in 24 cases for tuberculosis. It is interesting to note how nearly this corresponds with the experience of Sir John Thomson-Walker, who habitually adopted the conservative operation; he found that 8% of his cases subsequently required ureterectomy.

### III. *The Ureter Stump in the Presence of a Non-tuberculous Obstruction at the Lower End*

Kidd described in detail 12 cases demanding ureterectomy where the obstruction was due to a variety of causes other than tubercle. In infective conditions if no obstruction existed the ureter stump seldom gave trouble, and tended to sterilize itself. In the presence of a stricture attacks of colic may be caused or aggravated by the hypertrophying ureter muscle; an infected pouch becomes an empyema causing fever and pyuria. Peri-ureteritis and abscess may result. Dourmashkin called attention to the absence of symptoms other than pyuria, where a stone was impacted in a stump; one case, however, developed a ureteral and peri-ureteral abscess around a stone at the lumbar end of a stump twenty-five years after nephrectomy. Opinions based upon the experience of stone impacted in the lower end of the stump have shown considerable variation. No doubt the effects differ according to whether the stone causes complete or only partial obstruction. In his vast experience Marion was only twice forced to remove such a stone and thought ureterectomy unnecessary unless the stone were very large. The majority of surgeons, however, share the apprehension felt by Kidd, that the stone would act like a stricture causing empyema with its consequences. Where endoscopic or transvesical operations failed ureterectomy would probably be the wiser course, although a difficult undertaking. Hunt has found cases of this nature among the few indications for ureterectomy at the Mayo Clinic. I performed primary nephro-ureterectomy in the case illustrated in fig. 4 to avoid untoward consequences in the stump.

Troubles also occasionally arise from the stump in the following conditions:—

### IV. *Congenital Defects*

Hydro-ureter, with ureterocele and atresia of the ureteral meatus in extreme cases, may require ureterectomy if the ureter approaches the size of a coil of small intestine, as in two cases described by Gibson. Duplicated ureters, especially if their orifices are ectopic, are liable to great dilatation and to calculus formation, and should be similarly dealt with, especially if infected. I have had one such case and performed primary nephro-ureterectomy rather than heminephrectomy, on account of severe sepsis.

### V. *The Ureter with Incompetent Uretero-vesical Sphincter*

Hydro- or pyo-ureter in the presence of a lax ureter meatus may be of idiopathic or traumatic origin, and, unless the ureter is removed with the kidney, reflux with

ureteral fistula has been known to occur. Kimbrough has called attention to cases of incompetence of the uretero-vesical sphincter requiring the major operation. Plastic operations upon the meatus have been included in the aetiology, but I have not met with this condition either in these cases or after implantations of the ureter



FIG. 4.—Diffuse lithiasis, with calculi in the renal pelvis, upper and lower ureter. Marked ureteritis and peri-ureteritis at the lower end of the duct (woman aged 42). Juxta-vesical nephro-ureterectomy performed by the author.

following excisions of vesical tumours and diverticula. My personal cases of idiopathic ureteromegaly have been bilateral and outside the scope of surgery.

*Empyema of the ureter stump.*—This may be found whether obstruction exists at the lower end or not. Dourmashkin reported four and Hyman in detail three cases in the last 2 in. following subtotal ureterectomy (according to Papin's definition)

where the ureters were infected but not obstructed. The clinical course and the surgical treatment presented the same problems as are met with in infected bladder diverticula, persistent pyuria and cystitis being the main symptoms. Hyman found that excision of such stumps presents surgical difficulties so great as to induce the surgeon, when carrying out a primary ureterectomy for pyo-ureter, to excise the entire length down to its entrance into the bladder. As he says, a primary total ureterectomy may be quite difficult enough but the scar tissue of a former operation multiplies the problems disproportionately. Hence endoscopic measures, incision or dilatation of the orifice if there is a stricture, and a prolonged course of irrigation of the sac, should first be carried out in the hope of obviating the need for secondary excision, in accordance with the doctrines of Kidd and Fagerstrom.

#### PRE-OPERATIVE INVESTIGATIONS

*Clinical examination.*—After a full urine report the routine bedside examination is undertaken. A tender ureter, a thickened ureter, and sometimes a stone in the ureter will be apparent on abdominal palpation. Rectal and vaginal palpation are even more likely to give positive results.

*Cystoscopic inferences.*—The vesical pathology is studied. The ureter meatus may show characteristic changes. The passage of the ureteric catheter may reveal an obstruction at any level or produce excessive bleeding. If allowed, further passage of the catheter may tap clear urine, when blood was drawn at a lower level. This is the so-called sign of Chevassu and Mock, and is said to indicate a ureter papilloma. A check to the passage of the catheter is less often due to an organic obstruction than to spasms or kinks. Catheters or bougies of firmer consistence are often more informative than those of different sizes. Spasms may be overcome by anaesthesia, local or general, or belladonna and allied drugs.

While it is recognized that the drawn-up or "golf hole" ureter orifice indicates advanced tuberculosis at a higher level the reverse is certainly not the case, for I have in many instances found at operation very marked changes in the duct, even in the lower inches where cystoscopy had shown but little, if any, alteration of the orifice.

*Radiography* may show an altered renal outline, shadows in the renal or ureteric regions due to calculi and to the caseous deposits of tubercle.

*Pyelo-ureterography* by the ascending or the descending route defines most precisely the existing pathological states. The latter is often used to reinforce the former, except where tuberculosis is suspected, when the risk of dissemination cannot be ignored. The degrees and types of ureteromegaly can be well studied and note will be taken of the resiliency or rigidity of the tube, as to whether it has varied in size at different exposures, and the influence of such drugs as morphia, atropine, pituitrin, and especially eserine. The *diastolic* ureter if rigid indicates tuberculosis, the *resilient* ureter one *mechanically* distended from any of the causes already given, notably the systolic bladder. The tuberculous ureter offers a variety of appearances. Rigid ureteromegaly I have seen most often in children (fig. 5), but of course a degree of it, the "gas-pipe" ureter, is common enough in adults, no physiological systole ever being visible. In other cases there may be alternate areas of constriction or dilatation, with nowhere clearly defined edges—a worm-eaten appearance (fig. 6). The intensity of the ureterographic shadow depends upon the functional capacity of the kidney.

The ureterograph in papillomatosis would typically show areas of dilatation with filling defects placed according to the disposition of the growths. Such a ureter is better displayed by retrograde pyelography.

*Identification of the ureter stump as a focus of disease.*—This question frequently arises after nephrectomy for tuberculosis, when there is persistent cystitis and tubercle bacilluria. Exclusion of disease in the remaining kidney is accepted as incriminating the stump, but this is not always so, as a tuberculous bladder may not clear up even

when a descending source of infection is removed. It is clear that the problem would not have arisen had primary nephro-ureterectomy been adopted.

Apart from tubercle and papillomatosis, which have already been considered, recognition of the features of a diseased stump depends upon the clinical syndrome of an encysted focus of sepsis, rectal or vaginal palpation of a significant swelling,



FIG. 5.—Rigid megaloureter in a tuberculous ureter, from a boy aged 10.



FIG. 6.—The typical worm-eaten rigid tuberculous ureter of an adult. The right ureter is here affected, and the urogram demonstrates a cavernous focus in the corresponding kidney.

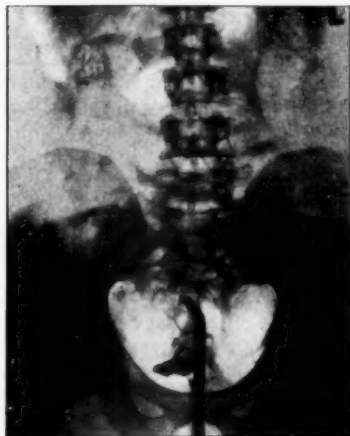


FIG. 7.—Demonstration of a ureter stump by passage of an opaque catheter through which sodium iodide solution was introduced.

persistent cystitis and pyuria with non-specific organisms. The cystoscopic appearance and the probing of the orifice with a ureteric catheter will determine whether it be stenosed or unduly patent. The best method of proof is to define the cavity with the aid of radiography, making use of the opaque catheter or, better, of an opaque medium introduced through it into the cavity (fig. 7). During the cystoscopic examination pus or blood may be seen to escape.

## THE DEVELOPMENT OF NEPHRO-URETERECTOMY.

The first removal of the kidney was carried out by Simon in 1869. Twenty-three years later, in 1892, Reynier successfully performed what is generally considered to be the first ureterectomy, removing an infected ureter responsible for severe symptoms after nephrectomy for pyonephrosis. This case was fully reported in Rousseau's Paris Thesis published in 1893. The following year Poncet (described in Riaudet's Thesis) also successfully excised the whole length of the ureter two and a half years after nephrectomy for tuberculosis, the ureter this time being removed to cure a tuberculous sinus which had remained since the nephrectomy, repeated curettings having failed to heal it. However, Papin refers to a uretero-nephrectomy performed by Pozzi in 1891, where the ureter was removed as a preliminary step, the proximal end being implanted into the skin of the loin; subsequently the kidney and upper segment of the ureter were removed successfully.

The first planned nephro-ureterectomy appears to have been performed by H. A. Kelly in 1893. In his original paper published in the *Johns Hopkins Hospital Bulletin*, February-March 1896, pp. 31-37, he gives full accounts of three cases upon whom he endeavoured to perform the complete operation. All were females suffering from renal and ureteric tuberculosis. With the aid of his cystoscope or bladder speculum he was able to prove the side by ureteral catheterization. In the first case a stout, otherwise healthy, girl, he removed the kidney and all but the last 3 in. of the ureter through a lumbo-ilio-inguinal incision, ligating the ureter posterior to the broad ligament. Two months later he tried to remove the ureter stump through the vaginal fornix, but found that adhesions and scar tissue could not be dealt with through the very constricted space available, and so failed to achieve his object. In his second case he removed the whole of the ureter through the abdominal incision, this time being helped by his assistant's finger which pushed up the vaginal vault and so made the ureter come within easier reach. In the third case, a stout female of 30, the same abdominal incision was made and the ureter was ligated at the broad ligament. The operation was completed bimanually, the fingers of the right hand pushed up the vaginal vault and made contact with the left hand, the vaginal wall alone intervening. The anterior fornix was then punctured with sharp scissors, and forceps, introduced through the hole so made, seized the ureter stump. The abdominal wound was closed and the ureter was dissected to the bladder through the vaginal incision, ligated, divided, and cauterized. The result in each case was successful.

Albarran in 1898 successfully performed nephro-ureterectomy for papillomatosis of the kidney, ureter, and bladder, also by the retroperitoneal route, in one sitting.

In spite of these successes it cannot be said that the operation of primary nephro-ureterectomy, at any rate for tuberculosis, gained acceptance, for in 1911 Lilienthal alluded to the strange unwillingness of most surgeons to dispose of the divided ureter after nephrectomy in a scientific and radical manner, and quoted the expressed opinions of several unnamed authorities, e.g. "The ureter seldom gives rise to any trouble even when diseased", "When infected the ureter should be stitched into the wound", and many others.

Having seen several examples, especially in tuberculosis, of lumbar sinuses and abscesses and retrovesical suppuration, he was convinced of the necessity for complete removal of the ureter, if this could be made rapid and easy. The method he devised was, after having removed the kidney, to pass a flexible urethral bougie down the ureter. This was then palpated and exposed extraperitoneally through a  $1\frac{1}{2}$  to 3 in. incision in the iliac fossa. The bougie was withdrawn through the loin and this allowed the ureter to be delivered through the fresh incision. He claimed that the ureter could then be easily traced by the finger down to the bladder, firmly ligated, divided, and disinfected with phenol. He mentioned the risk of ulceration of the

great vessels from the pressure of the drainage tube, unavoidable with this incision for exposure of the ureter.

It is noteworthy that the two-way abdominal approach of Lilienthal introduces the modern method of whole-length exposure of the kidney and ureter by combined, but separate, incisions. Hitherto the single long incision used by Kelly, Albarran and Israel was standardized, with sporadic appearances in the literature of novel methods for approaching the vesical end; Kelly's vaginal method has been mentioned—others tried an approach through the perineum with or without the additional exposure gained by removal of the coccyx or part of the sacrum, somewhat similar to the Kraske approach to the rectum. Needless to say these methods found little favour. The over-long lumbo-ilio-inguinal incision jeopardized the musculature and no doubt was responsible for many herniæ. These deficiencies in surgical technique evidently partly explain the lag in progress of ureteral surgery, so that Lilienthal's method, designed for preservation of the abdominal wall, marked a valuable advance.

In 1913 Kidd suggested a modification of the lower or inguinal incision for exploring the pelvic ureter, also with the object of conserving the abdominal musculature. It was horizontally placed, 3 in. in length, of which the inner third lay over the rectus muscle and the central point of the outer two-thirds immediately above the internal abdominal ring. This incision allowed internal retraction of the rectus muscle; the muscular and tendinous structures of the outer two-thirds were split in the direction of their fibres. Kidd claimed that he could approach the pelvic ureter satisfactorily in this way. Swift Joly, commenting upon this method a week or two later, stated that he had found a vertical hypogastric incision, extending from the umbilicus to the symphysis pubis, to give a very adequate approach to the base of the bladder and to either or both ureters, as might be required for removal of stones or for palpation of thickening of either ureter as an aid to the recognition of the side of a tuberculous focus. This appears to be the first allusion to a vertical mid-line incision, so that Joly's name might justifiably be attached to it. The following year Judd advocated the same incision, observing that he was not aware that it had been employed previously. One of the advantages he named was the better opportunity it offered for elevating the bladder and so raising the intramural section of the ureter when the peritoneum was separated to allow of easier mobilization. The majority of urological surgeons have adopted this incision. Others have preferred to use an oblique inguinal incision either of the muscle-splitting "grid-iron" type, or one similar to the Astley Cooper or Abernethy incision for ligature of the external iliac artery. A rectus-splitting or pararectal incision is also frequently employed, and either is of especial advantage in a secondary ureterectomy, where some length of the abdominal ureter additionally requires removal. The high Trendelenburg position is necessary whichever of these incisions is chosen.

*The present-day method of nephro-ureterectomy* evolved out of trial and error has shown no striking developments in recent years, merely improvements in details of surgical technique, notably the "extraperitonization" method of Papin to which I shall allude.

The majority of surgeons prefer to remove the kidney first, its exposure may reveal a ureter less diseased than suspected and whose entire removal is obviously unnecessary. It is easier to curtail the operation if surgical shock demands. There is less risk of wound infection if the opened ureter is free in the wound momentarily, for in nephro-ureterectomy division of the ureter is the last step. The most difficult part of the operation, the separation of the ureter from the bladder, is easier in the later stages of the operation with better muscle relaxation. Finally, it is simpler and quicker to transfer the patient on the table from the lateral to the dorsal posture, than vice versa. Gutierrez is one of the few advocates of the reverse operation, mainly on the grounds that the severed ureter may be used as a guide to the vascular pedicle and so facilitate separation and removal of a large adherent kidney.

In my first three radical operations for tubercle I removed the ureter first, rather by accident. My first case was a mistaken diagnosis. I exposed the vesical end of the ureter expecting to find a stone. The true condition, namely tuberculosis, was at once obvious, and so I continued the radical removal as a uretero-nephrectomy. The satisfactory outcome encouraged me to do my next two cases in similar fashion. These early cases were before the introduction of excretion urography, and in the first case, where impacted stone was suspected, retrograde pyelography was prevented by inability to pass a ureteric catheter. The sounder arguments in favour of removal of the kidney first influenced me to reverse my procedure.

Of the various operations for removal of the ureter supplemental to nephrectomy, choice will be made from the following methods :—

I.—*Nephro-ureterectomy.*

(1) *Primary one-piece operation.*

(2) *Primary two-piece operation* (deferred ureterectomy; this is a two-stage primary nephro-ureterectomy).

(3) *Secondary ureterectomy*, i.e. late ureterectomy, performed in consequence of persistence or development of indicative symptoms.

II.—*Uretero-nephrectomy.*

*Definition of ureterectomy.*—According to Papin, radical or “total” ureterectomy must include the intramural section; thus to complete the operation a disc of bladder wall surrounding it must be removed, as may be required in certain cases of tuberculosis and neoplasm. “Juxta-vesical” ureterectomy indicates removal flush with the bladder. “Subtotal ureterectomy” is understood by Papin, and may be accepted to mean removal in the male to the point where the duct is crossed by the vas deferens, in the female to the base of the broad ligament, or, more precisely, to the point where it lies below the uterine artery, called by Papin “the uterine pedicle”. More limited removal Papin calls “partial” ureterectomy.

The titles of the various operations indicate their nature; thus the primary one-piece operation is self-explanatory. Deferred ureterectomy is the method adopted when it is considered that the whole operation at one sitting would be beyond the capacity of the patient's endurance. The kidney and ureter to the pelvic brim are removed at the first stage, the upper end of the stump being freed, as far as possible, from the great vessels at the pelvic brim and displaced downwards in order to render easier the securing of the upper extremity, one of the difficulties of a subsequent ureterectomy. An example of the necessity of this operation is seen in a case of diffuse papillomatosis of the kidney, ureter, and bladder which I have already cited, and represented in fig. 2.

I. *Technique of Extraperitoneal Nephro-ureterectomy.*

*Step 1.*—The kidney is exposed through a curved lumbar incision which extends from over the last rib at the outer border of the erector spinae to usually 2 in. above and behind the anterior superior iliac spine (fig. 8). The vascular pedicle is ligated and the ureter is freed as low as the incision permits, i.e. usually just below the pelvic brim. The kidney is left suspended from the lowest part of the wound by the ureter, and the muscles and skin are sutured. The lowest sutures of muscle and skin are emplaced, but not tied until after the kidney is removed, the space temporarily left may be of value in separating anchoring adhesions overlooked in the initial stages of the operation (fig. 9).

*Step 2. Approach to the pelvic ureter.*—I prefer Joly's vertical incision half an inch to one or other side of the mid-line, as I believe a better scar results than from one

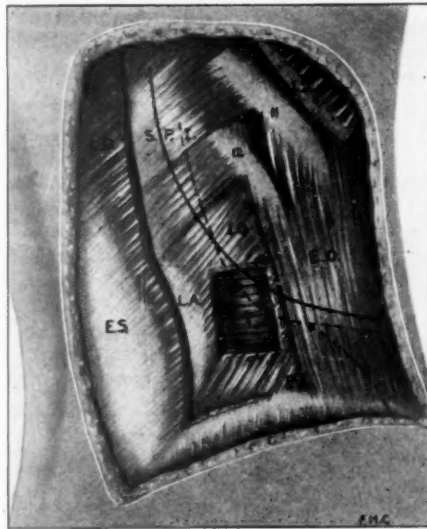


FIG. 8.—Semi-diagrammatic representation of muscle strata, &c., concerned in exposure of the kidney through the curved lumbar incision adopted by the author. A broad window in the latissimus dorsi (L.D.) exposes the lowest ribs (11 and 12), the serratus posticus inferior (S.P.I.), the external oblique (E.O.), the internal oblique (I.O.), and lumbar aponeurosis (L.A.). A window in the internal oblique shows the transversalis (T.). The erector spinæ (E.S.) is seen through the fibres of the latissimus dorsi. P. represents the position of Petit's triangle. The incision crosses the course of the last dorsal nerve (broken line) which, when seen, is displaced downwards, the approach to the kidney being effected above the nerve.

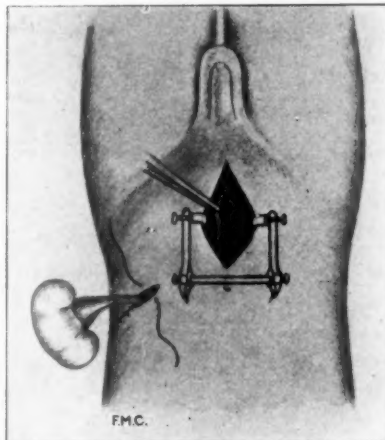


FIG. 9.—The pelvic ureter is exposed through the incision originally recommended by J. Swift Joly. The lowest deep and superficial sutures of the lumbar incision are emplaced but not tied until the kidney and ureter are removed.

in the linea-alba (fig. 10). If there is difficulty in separating the ureter in its lower abdominal course I make my incision still more lateral so that it can be continued by splitting the rectus to above the umbilicus if necessary, thus giving opportunity for better exposure of the ureter at and above the pelvic brim. I also prefer to make the incision at least  $\frac{3}{4}$  in. from the mid-line in stout patients, and in a secondary ureterectomy where the length of the ureter stump is doubtful.

*Anatomy of the pelvic ureter.*—The extremities of the pelvic ureter are fixed points and present no great difficulty in exposure, with the aid of the well-known anatomical landmarks, and provided that plenty of room is permitted by the adequacy of surface

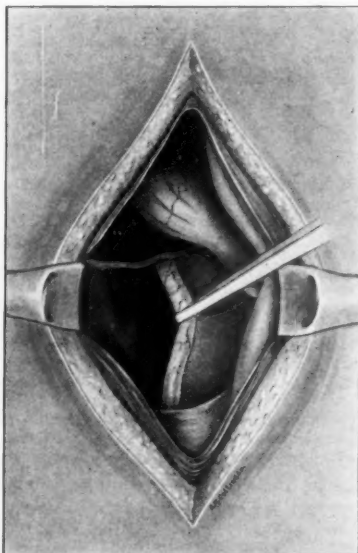


FIG. 10.—Separation of the lower third of the ureter. The superior vesical artery has been divided and ligated to allow displacement and elevation of the bladder. The relation to the vas and great vessels is seen. The vertical Joly incision is used and the ureter is supported on a gauze sling.

approach. The course of the duct, since it is attached to the peritoneum, must vary with the position of the peritoneum, which lies loosely on its fatty bed and shifts according to the degree of distension and movements of the bowel and bladder. A distended ureter increases in length as well as in calibre and, therefore, it becomes tortuous to be accommodated in the space available when its recognition may, at first, be difficult, especially if the walls have become stretched and thin. If there is no thickening or impacted stone as a guide, it may be necessary to pick and up roll the peritoneum between the finger and thumb to aid in the search. Peritoneum held by a retractor may shift the ureter far from its normal position; it may be drawn up to the anterior abdominal wall or forced into the depth of the wound. Post-operative scar tissue, uterine or ovarian tumours and fat add to the difficulties of exposure.

When the ureter reaches the pelvic floor its forward course bears important relations :—

(a) *In the male.*—(i) The *vas deferens* rising up from the prostatic region mesial to the ureter arches over it passing in an upward and outward course. It crosses the ureter about  $1\frac{1}{2}$  in. above the entrance to the bladder. Kidd describes the relations of the vesical arteries and the vas to the ureter as comparable to the position of the fingers and thumb holding a pen preparatory to writing, the pen representing the ureter, the fingers the vesical arteries, and the thumb the vas. The pen is held in the right hand in the case of the right ureter.

(ii) *Arteries.*—The termination or terminal branches of the internal iliac lie postero-external, and the vesical arteries to reach the bladder cross the ureter anteriorly from without inwards. The superior and middle vesical arteries are branches of the hypogastric and the relation of these to the ureter is variable. The

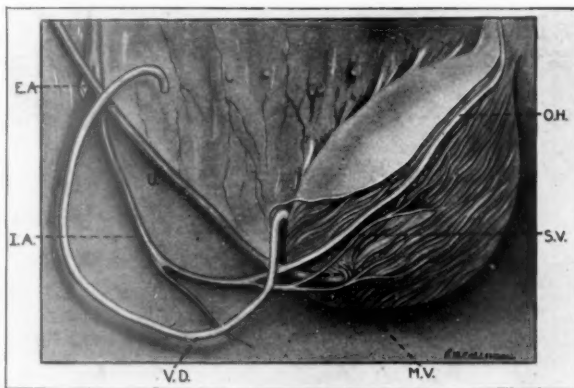


FIG. 11.—Relations of the pelvic ureter in the male. There are shown the crossing of the ureter (U.) by the vas (V.D.), the superior and middle vesical arteries (S.V. and M.V.), and obliterated hypogastric (O.H.). The vas has been divided. The area of peritoneal attachment to the bladder is seen. I.A. and E.A., internal and external iliac arteries.

[Drawn for the author from Spec. No. 291, Room No. 2, Hunterian Collection, R.C.S. Museum, with the permission of the Royal College of Surgeons.]

three vessels may cross the ureter independently or in a common stem, the vesical arteries not leaving the unobliterated section of the hypogastric until after the latter has crossed the ureter (fig. 11).

(b) *In the female.*—In separating peritoneum from the lateral wall of the pelvis the round ligament will at once stand out prominently, its firm attachment at the internal ring and its fixation to the peritoneum preventing mobilization of the serous sac. (Division of this ligament simplifies the subsequent procedures and seems to give no resultant disability.) The trans-pelvic course of the ureter differs in no respect from that in the male until the broad ligament is reached. Here there is the relationship of the uterine vessels (fig. 12). The uterine artery has a long course from its origin to its disappearance into the base of the broad ligament, and it may be torn if too much traction is made in displacing the peritoneal sac towards the opposite side.

In addition to the uterine, vesical arteries, and those to the ureter itself, a plexus of veins lies in the fatty cellular tissue at the base of the broad ligament, and actually forms a venous ring around the ureter which drains into an anterior and a posterior trunk passing up in front of and behind the ureter to the iliac vein.

The ureter passes obliquely forwards and inwards in the base of the broad ligament half-way between the pelvic wall and the cervix, usually about 2 cm. from the latter. The uterine artery runs transversely inwards and is therefore posterior to the ureter opposite the cervix.

*Step 3. Extirpation of the pelvic ureter.*—The high Trendelenburg posture having been arranged the incision is deepened to expose the peritoneum, and this is at once peeled off the lateral pelvic wall commencing below, where it is more easily mobilized. When working up to the pelvic brim the vas or round ligament hinders its upward separation and a firm attachment of the sac near the anterior iliac spine may require

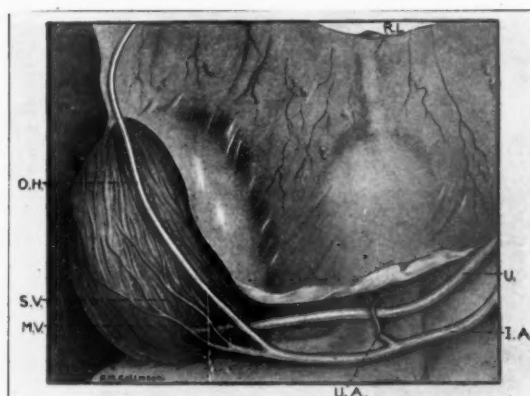


FIG. 12.—Relations of the pelvic ureter in the female. The crossing of the ureter (U.) by the uterine artery (U.A.) as it enters the base of the broad ligament is seen and, more anteriorly, are demonstrated the relations of the obliterated hypogastric and vesical arteries (O.H., S.V. and M.V.). The bladder is artificially displaced forward in this specimen. I.A., internal iliac artery.

[Drawn for the author from Spec. No. 296, Room No. 2, Hunterian Collection, R.C.S. Museum, with the permission of the Royal College of Surgeons.]

division with scissors, at the same time care being taken not to cut too deeply, or the peritoneum may be opened and adherent bowel damaged. The vas is separated from the sac, but it is usually necessary to divide the round ligament. Separation of adherent peritoneum will be simplified by passing the hand into the iliac fossa, tracing on the sac downwards and lifting it off the pelvic brim and vessels. The peritoneal attachment in the lower and outer iliac region will then be reduced to a narrow process which may be divided with greater surety. Whilst proceeding in this way the ureter will soon be recognized, and more easily if its tension is altered by traction on the kidney. While separating the ureter the kidney should be lifted to relax it, and a gauze sling is passed under it and held in forceps. All strands of tissue passing to the ureter should be double clamped and ligated, as each may contain a vessel which if carelessly torn is prone to continue to bleed in the depth of the fatty bed, or the bleeding point may be a vessel of considerable size torn close to its parent iliac vessel. The ureter is usually separated without much difficulty to the point

where the vas arches over it in the male and to the base of the broad ligament in the female. In many cases the operation will have been regarded as fundamentally completed if the ureter is divided at this level. Papin regards it as impossible for the terminal section to be removed if only the lumbo-ilio-inguinal incision be used; a vertical hypogastric incision is a necessity. Both Judd and Papin, advocating this incision, have described the next important step, namely elevation of the bladder by means of which the intramural region is raised and more completely exposed. Judd dissected the peritoneum from the superior and posterior walls. Papin went a stage further advising that the disc of peritoneum some 6 cm. in diameter, which is firmly welded to the bladder in the region of the urachus, should be detached from the peritoneal sac. To effect this the peritoneum is opened at the edge of the attached

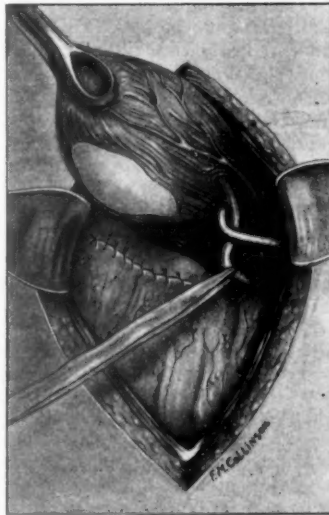


FIG. 13.—To gain better approach to the vesical end of the ureter the bladder has been mobilized by Papin's method of "extraperitonization". There are seen the elevated bladder with peritoneal disc attached, the relation of the vessels and vas to the ureter (supported on sling) and the sutured gap in the serous sac.

section near the urachus and this edge is followed with scissors in a circular plan. The gap in the peritoneum so left is closed with a continuous suture. The disc of peritoneum is mainly situated on the superior wall, for separation of the serous coat from the posterior wall of the bladder is always easy. There is no doubt that this manœuvre is a considerable asset and I have found it actually an economy of time, for it facilitates hæmostasis and undoubtedly provides a means of a more satisfactory approach to the intramural region (fig. 13). So far I have only on one occasion had to remove the intramural ureter since adopting it. In either sex the bladder is held up in forceps or by a suspending suture and the ureter is traced down while supported on its gauze sling. The peritoneum with its contents is retracted upwards and laterally. All strands which may be vascular structures, notably the uterine, superior, and middle vesical arteries, are divided between clamps. This will allow rotation and elevation of the bladder and at the same time more complete exposure of the ureter, which is drawn upon and separated until a knuckle of bladder comes into view.

In the female, division of the uterine artery will allow retraction of the cervix and, indeed, of all the broad ligament structures, as though a pedicle were divided, so justifying Papin's reference to the uterine artery as the "uterine pedicle".

Alternatively to dividing the uterine artery the ureter may, after separation from the bladder, be dissected and freed below the artery or divided above the artery, and the residual stump is then removed separately from below (anterior to) the artery.

*Step 4. Separation of the ureter.*—(i) In the majority of cases it suffices to divide it between double clamps, to treat its ends with pure carbolic and to ligature either end. The lower clamp lies flush with the bladder. Some prefer to divide with the actual or diathermy cautery. The lower cut end may be buried by a Lembert or purse-string type of suture carried through the bladder wall, but such burying does not seem to be necessary and is rather difficult to carry out.

(ii) Where the intramural ureter is the seat of papillomata it must be removed by encircling it with a diathermy knife, thus removing a collar or disc of surrounding bladder wall. Some have advised a similar procedure in tuberculosis, but this could scarcely satisfy the ideal of a "closed" operation, for it would be difficult to avoid soiling of the wound with vesical contents either at operation or subsequently, an unhealthy state of the bladder predisposing to separation of the sutured edges of the bladder wound, as happened in one of my cases, resulting in the breaking down of the wound.

(iii) Fulguration of the intramural canal has been recommended by J. C. Colston, where papilloma transplants may exist (*see supra*). An electrode is introduced before the lower end is ligated. The cautery point is worked up and down so that all the mucous surface may be treated; the stump is then ligated or sewn. Colston advised that there be some fluid in the bladder so that the cautery can damage no part of the wall.

*Step 5. Nephro-uretero-cystectomy.*—All the preliminary stages completed, the bladder is opened anteriorly and its base illuminated with one of the modern retractors of the Morson or Bernard Ward type. A growth situated near the ureter orifice is surrounded by an encircling incision made with a diathermy knife. The excised section is removed in continuity with the ureter and the resultant opening closed with interrupted sutures. It is better to drain the bladder suprapubically than with an indwelling catheter.

Whichever of these methods has been employed all that remains is to draw upon the kidney and to separate any few remaining adhesions. The hypogastric incision is closed, allowing for ample drainage. The lower sutures of the upper wound are tied. No drainage to the loin is necessary.

## II. Technique of Uretero-nephrectomy.

The Trendelenburg posture is adopted, the ureter is traced to the bladder and separated in the manner to be described, double-clamped, divided, cauterized, and ligated. The upper end is traced and freed as high into the iliac fossa as possible. The hypogastric wound is sutured and drained to the retrovesical space. The patient is then shifted to the lateral posture and the kidney is exposed from the loin. The lax ureter is at once sought and forms a ready guide to the vascular pedicle. The pedicle is separated, clamped, ligated and divided, allowing removal of the kidney and ureter in one piece. A loin drain is unnecessary.

## POST-OPERATIVE COMPLICATIONS AND RESULTS

The complications have been few, and with the exception of the one case of "total" nephro-ureterectomy for tubercle, where the intramural ureter was removed, the average stay in hospital was 21.5 days, actually a day less than after nephrectomy alone! In the exception referred to, inefficient post-operative bladder catheter drainage was responsible for an over-distended bladder with consequent yielding of

the sutured site of the ureter. A loin fistula took two months to heal, and then only with the aid of suprapubic cystostomy. This was the only major complication in my series of 30 cases, 24 of which were performed for tubercle and the remainder for growths, stones, and developmental defects.

Of the *tuberculous* cases, the operations were :—

Total nephro-ureterectomy ..	1
Juxta-vesical ureterectomy ..	17
Uretero-nephrectomy ..	3
Secondary ureterectomy ..	3
	<hr/>
	24

There was no operative mortality in the total series and I am aware of only two late deaths amongst the 30 cases, one, tuberculous, who died three years after in uræmia probably from tuberculosis of the second kidney, a kidney always suspected but never proven tuberculous. The other death was the case of primary papillomatosis of the ureter who died three years later apparently from a growth in the liver; there was no recurrence of papillomata.

During the period of these operations, approximately twelve years, I also carried out 50 nephrectomies with partial ureterectomy for tubercle. The low number of my late ureterectomies must at once raise the question: "Why perform the major operation when, after the minor, ureterectomy is so rarely needed?" The answer is, partly, that by primary removal of 30% of ureters in the cases presented for operation, i.e. 21 ureterectomies in 71 cases, the need was anticipated and therefore excluded. But I cannot say that the remaining 70% were satisfactory for, of the 50 cases, 23 could not be traced, and of the rest, three had unhealed sinuses in the loin for many months, others tubercle bacilluria and persistent cystitis, and several extra-urinary foci which denied further urological investigations.

It is clear that the final answer to the question of the relative merits of the major and the minor operation in tuberculosis can only be on the evidence of many hundreds of both operations, and not until the subjects have been followed through a period of, at least, a decade. Bad as the ureter may often seem at the primary exploration of the kidney, it is phenomenal to what extent and with what rapidity recovery may take place, as proved symptomatically, bacteriologically and, in a few cases, where opportunity has arisen for direct examination of the ureter at autopsy or when some other operation has been required. On the other hand, where the outcome has not been so good and the reduced constitutional state, involvement of other structures, or the refusal to submit to further operation now deny it him, the surgeon may have cause to regret a lost opportunity.

It is no longer an axiom in the surgical treatment of tuberculosis to remove all possible foci, but where an operation, agreed by all to be necessary (nephrectomy) can be extended without severe risk, it should include a useless anatomical structure which, in all probability, is a potent source of infection. On that point, however, no surgeon at the time of his operation can be quite certain; there is no bell that rings to tell us definitely that here is a ureter which will or will not recover. Is it not wiser then to remove it?

Whilst, therefore, the verdict in the case for primary nephro-ureterectomy in the presence of renal tuberculosis must be regarded as open, a submission in favour of this operation is reasonable if it appears to be within the capacity of the patient's endurance, especially if he is young. As age advances greater technical difficulties are met. Stoutness presents a considerable hazard, scar tissue from other operations, and the greater complexity of the pelvic vascular system, especially in the multipara of advancing years, introduce obstacles which will limit the freedom of choice of operation. Fortunately renal tuberculosis of the middle-aged, and aged, is prone to run a milder course, and conservatism in its surgery is the more permissible. In

adolescence and young adult life, and up to the age of 35, preference should be given to juxta-vesical nephro-ureterectomy.

My object has not been to urge primary nephro-ureterectomy in tubercle or, indeed, in any other cases than neoplasm. It has been rather to call attention to some noteworthy developments, especially in operative technique. In a patient offering a fair operative risk the danger of the major operation is little if any greater, convalescence is not prolonged, and the prospects of recovery are materially improved. To have performed total ureterectomy will insure the surgeon against one of the more unpalatable problems to be faced in urology: "Shall I go for that ureter stump?"

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*Discussion.*—Mr. HUGH LETT proposed a vote of thanks to the President for his valuable address.

Mr. JOCELYN SWAN, in seconding the vote of thanks to the President, congratulated him upon his lecture, the value of which was so much enhanced by the fact that he was able to include such an amount of material from his own personal observation. With regard to the removal of the ureter, Mr. Swan was strongly in agreement with the President that this should be carried out, unless special circumstances prevented, in all cases in which nephrectomy was performed for tuberculous disease. Preferably the operation should be carried out by first separating the kidney and ligating the renal vessels, leaving the renal pelvis and upper ureter intact, through a small lumbar incision. The kidney is then tucked into a space made in the retroperitoneal tissues by separating the peritoneum downwards towards the iliac fossa and the lumbar incision is sutured. A second incision is then made in the iliac area and the peritoneum separated inwards, when the kidney and intact ureter are easily delivered from the incision. The ureter is then separated from the peritoneum downwards to the retrovesical space and clamped and ligated close to the bladder. The only difficulty likely to be met with is in the final separation of the ureter from under the broad ligament in the female, when bleeding may arise from the uterine veins. Mr. Swan had seen several cases in which a tuberculous fistula had resulted in cases in which the infected ureter had not been removed with the kidney and he could see no reason why a length of infected ureter should be left to remain a potential source of infection.

When operating for a papilliferous tumour of the renal pelvis, Mr. Swan expressed the opinion that not only should the kidney and ureter be removed, but that the terminal intramural portion of the ureter, together with the immediate vesical wall, should be resected, though he did not go to this length in the removal of tuberculous disease.

## Section of Radiology

President—PETER KERLEY, M.D.

[January 18, 1940]

### DISCUSSION ON CHOLECYSTITIS

**Mr. Robert McWhirter:** In cholecystography special attention should be paid to technique so that technical errors are eliminated, and reports should be standardized. By informing the clinicians periodically of the percentage accuracy in each group the significance of these standard reports will come to be appreciated.

*Technique.*—The technique used in Edinburgh Royal Infirmary is similar to that of the Mayo Clinic, but a few minor modifications have been made. A straight film of the gall-bladder region is taken in the first place and, if gall-stones are found, investigation by means of sodium-tetraiodophenolphthalein is only rarely carried out. Prior to the evening meal 2 gr. of phenobarbital are given to lessen the possibility of sickness after the ingestion of S.T.I.P.P. Sickness, in addition to leading to loss of dye, tends to increase the amount of gas in the bowel. The enema on the morning of the examination has been discontinued because it was found that, frequently as a result of faulty technique, the amount of gas in the colon was actually increased instead of being diminished.

Let us suppose for example that the patient is to be examined on a Wednesday, these instructions are given him :—

- |           |  |
|-----------|--|
| Monday    | (1) Take a purgative at night-time.  |
| Tuesday   | (2) Take the two tablets provided (phenobarbital gr. ij) at 6 p.m.   |
|           | (3) Take a full meal at 7 p.m. but omit cream, butter, eggs, and other fats.   |
|           | (4) <i>Immediately</i> after this meal empty the entire contents of the bottle (opacol) into a glassful of water, stir until the blue solution becomes white, and drink all of it. |
|           | (5) Do not take any further laxative. You may drink water, black coffee, or clear tea.   |
| Wednesday | (6) Do not eat breakfast. Come to the X-ray Department a little before 10 a.m.   |

Films are taken at 10 a.m. (i.e. the 14th hour) and may be taken thereafter at two-hourly intervals up to the 20th hour. But as soon as the gall-bladder shadow becomes of good density, a fat meal is given and a film taken one hour after this.

The patient should be placed comfortably in the prone position on a flat-topped table equipped with a synchronized Bucky. The exposure, made in normal expiration, should not be longer than half a second. Prior to the exposure being made the

patient should be instructed to take a little breath in, and not a deep breath, as the abdominal organs after a deep breath take some considerable time to reach a stationary position again. Compression is required in all cases and should be made with the compression band over the lower ribs. A pad of cotton-wool and oblique views are generally successful in disposing of overlying bowel shadows.

It will be noted that the 7 o'clock meal is fat free. The presence of fat delays the emptying of the stomach, and the prolonged flow of fatty food into the duodenum interferes with the concentration of the dye. It has also been shown that the presence of fat in the bowel renders a greater amount of the dye insoluble.

No large film is taken of the whole abdomen because this would appear to be unnecessary. It is customary in some centres to do this, in the belief that the examination is of little value if much dye remains unabsorbed in the colon. Obviously, however, when the gall-bladder is so diseased that it cannot concentrate it, the dye cannot simply vanish. One would therefore expect in such cases to find a considerable quantity in the colon.

The incidence of vomiting is reduced by giving phenobarbital and by giving the dye immediately after the 7 o'clock meal. Satisfactory shadows may be obtained even when the patient is sick very soon after taking the dye, but if a poor shadow is found, or if the gall-bladder cannot be visualized, the examination should be repeated.

The differential diagnosis of shadows in the right upper abdomen is as a rule a very simple matter. An oblique film with the right side of the abdomen slightly raised from the table is generally all that is required. In this way one can distinguish calcification of the costal cartilages and renal calculi. Gas shadows vary in shape and position and pancreatic calculi are so rare that they scarcely deserve mention.

*Film interpretation and film reporting.*—It should be realized clearly that the report on gall-bladder films, following the administration of S.T.I.P.P., is a report on the function of the organ and so in some respects is quite different from any other radiological report. Occasionally gall-stones, not previously visible, may be seen by means of the dye, but the determination of the function of the gall-bladder is the most important part of the examination. It is surprising, therefore, to find that in many hospitals the statement of the function is essentially a matter of personal judgment. This personal assessment is expressed in a multitude of ways and naturally varies according to the member of the radiological staff who happens to report the films. Surely the duty of an X-ray department is not merely to issue reports which *may have* some meaning for the particular radiologist responsible, but to issue reports, the significance of which will be appreciated in the wards. If the radiologist is indefinite and if his opinion is accepted as the best available, then a proportion of the examinations will be inconclusive.

All degrees of density of the gall-bladder are to be found, from complete absence to a shadow of excellent density. The absence of a shadow or a very faint shadow indicates disease, while a good shadow suggests that the gall-bladder is healthy. The difficulty lies in drawing a line of demarcation between the faint shadow and the shadow which is to be regarded as of normal density. The following figures will show that comparison of the gall-bladder density with the shadow of the liver enables such a line of demarcation to be drawn with reasonable accuracy and that this comparison also permits of reports being classified into three categories, whilst the personal factor is in the main eliminated.

The three standard reports suggested are : (a) Normally functioning gall-bladder. (b) Poorly functioning gall-bladder. (c) Non-functioning gall-bladder.

In each case the report should indicate the presence or absence of gall-stones.

A report that the gall-bladder is normally functioning indicates that the density of the gall-bladder shadow is greater than that of the liver, while poorly functioning gall-bladders have a density less than that of the liver. A report that the gall-bladder is non-functioning is self-explanatory.

The selection of the liver shadow has the advantage that it takes into account the stoutness of the patient—a factor which obviously influences the degree of density of the gall-bladder image. Comparison with the shadows cast by the ribs and spine is unsatisfactory, for these structures may vary in density as the result of factors other than those associated with the build of the patient.

If a faint gall-bladder shadow is superimposed on the liver its density will be accentuated just in the same way as the shadow cast by the kidney is increased when the margin of the liver overlaps this organ. It is necessary, therefore, to select a portion of the gall-bladder shadow which is not superimposed on the liver. This area should then be compared with the densest part of the liver shadow by masking out the surrounding portions of the film. It is surprising how easy it is to delude oneself that the gall-bladder shadow is denser than the liver if masking is not adopted.

It is suggested that any further sub-grouping will only lead to confusion, and in any case there is no other suitable shadow which will serve as a standard in separating such sub-groups. Accordingly, if the above suggestions are adopted, there need be no other reports than the above three. The matter will be discussed again after the results of this system of reporting have been considered.

#### *Operative and autopsy results.—*

*Cholecystographic Data for 1932*  
(Mayo Clinic Series, *Brit. J. of Surg.*, 23, 155.)

Diagnosis	Cases	Findings at Operation								Diagnosis confirmed at operation Per cent.
		Gall-stones	Tumours	Cholecystitis—Grade					Normal	
				Not graded	2	3	4	Miscellaneous		
Normally functioning gall-bladder	287	4	2	19	4		1		257	89.5
Poorly functioning gall-bladder	39	28 (71.8%)		5		1	2	1	2	94.8
Non-functioning gall-bladder	106	90 (84.9%)	3	4	3		2	1	3	97.1
Normally functioning gall-bladder with stones, 124										
Poorly functioning gall-bladder with stones, 78	294	293	1							99.6
Non-functioning gall-bladder with stones, 92										
Tumour .. .. .	6		6							100.0
Total ..	732	415	12	28	7	1	5	2	262	

445 cases with positive cholecystographic data ; 6 errors (98.6% correct). Of 732 diagnoses, 696 (95.0%) were confirmed.

415 cases with gall-stones at operation ; 411 (99.0%) with positive cholecystographic data. Of the 415 cases, gall-stones were visualized and reported in 294 (70.8%).

470 cases with disease of the gall-bladder at operation ; positive cholecystographic data in 440 (93.6%).

262 cases without disease of the gall-bladder at operation ; negative cholecystographic data in 257 (98.0%).

*Cholecystographic Data for a Three-Year Period  
(Edinburgh Series)*

Diagnosis	No. of cases	Findings at Operation or Autopsy					Diagnosis confirmed Per cent.
		Gall-stones	Cholecystitis and stones	Cholecystitis	Miscellaneous	Normal	
Normally functioning gall-bladder	34	1	4	2	0	27	79
Poorly functioning gall-bladder	24	2	8	12	2	0	100
Non-functioning gall-bladder ..	127	29	59	21	14	4	97
Normally functioning gall-bladder with stones	15	9	5	0	1 (s)	0	100
Poorly functioning gall-bladder with stones	17	3	13	0	1 (s)	0	100
Non-functioning gall-bladder with stones	34	13	21	0	0	0	100
Total .. ..	251	57	110	35	18 (2s)	31	

217 cases with positive cholecystographic data; 4 errors (98% correct). Of 251 diagnoses, 240 (96%) were confirmed.

169 cases with gall-stones at operation; 164 (97%) with positive cholecystographic data. Of the 169 cases, gall-stones were visualized and reported in 66 (39%).

220 cases with disease present at operation; positive cholecystographic data in 213 (97%).

31 cases without disease at operation; negative cholecystographic data in 27 (87%).

The Edinburgh figures are comparatively small. They extend over a period of three years and in this time approximately one thousand cholecystograms were made.

There are two points worthy of consideration when the two sets of figures are being compared. Firstly, in the Edinburgh series only 79% of the normally functioning gall-bladders were confirmed by operation to be free of disease, while in the Mayo Clinic series, 89% were confirmed. Secondly, the proportion of cases in which gall-stones were visualized is much smaller in the Edinburgh series, the figures being 39% as compared with 71%. It would be only fair to state, however, that in the former instance the difference may be mainly accounted for by the fact that only a very small proportion of the normally functioning group were operated on. Of this number the clinical evidence probably suggested gall-bladder disease.

*Cholecystographic Data 1936 to 1939*

Diagnosis	Number of reports issued	Operation or autopsy reports received	Percentage of cases in which reports were received
Normally functioning gall-bladder ..	530 (app.)	34	6
Normally functioning gall-bladder with stones	40 (app.)	15	38
Poorly functioning gall-bladder with or without stones	80 (app.)	41	51
Non-functioning gall-bladder with or without stones	350 (app.)	161	46
Total .. ..	1,000 (app.)	251	25

Again in Edinburgh we do not, unfortunately, obtain the same full operative notes as in the Mayo Clinic. In cases, other than those indicated, the patient had an abdominal operation but no mention is made of the state of the gall-bladder which, though presumably normal, cannot be stated to have been confirmed as normal.

As in most hospitals we can usually rely on hearing of our errors but not necessarily of our successes. The much smaller proportion of gall-stones visualized before operation in the Edinburgh series is accounted for by the fact that if gall-stones are found in the preliminary film the case does not usually proceed to cholecystography and only the series investigated by S.T.I.P.P. have been considered in this paper.

Apart from those two exceptions it will be noted that the figures are essentially similar, and the following points emerge on comparison of the operative or autopsy findings with the method of X-ray interpretation.

If no shadow is cast by the gall-bladder, or if its density is less than that of the liver, disease is present in 98% of cases in both series of figures. The accuracy thus achieved is definitely beyond that obtainable by clinical means, and the value of the examination is demonstrated.

In the normally functioning group the value of the method is less obvious. It should be mentioned here, however, that if there is doubt as to whether a gall-bladder is normally functioning or poorly functioning it is placed in the normally functioning group. This method of reporting enables the clinician to infer that disease is almost certainly present if the gall-bladder is reported to be poorly functioning. It might be argued from this and from the operative findings, that a further sub-group should be made to include those cases which are just denser than the liver shadow. On going into the matter, however, it is found that while such a sub-group may represent disease in a proportion of cases, errors also occur when the shadow is of excellent density. Accordingly, it follows that all the healthy gall-bladder cases cannot be separated out by means of cholecystography and, in any case, as already mentioned, there exists no clear distinction between this sub-group and the group with good shadows. The personal factor would again become prominent.

A further point to be considered in the findings of the normally functioning group is that a gall-bladder may retain the power of concentrating the dye satisfactorily when it is partly diseased. In the majority of these cases there is only early cholecystitis present. While the operative findings must be the basis of checking X-ray reports, it does not necessarily follow that the patient will obtain complete freedom from his symptoms following cholecystectomy for a gall-bladder showing some evidence of disease and yet capable of normal concentration.

In the poorly and non-functioning groups all such cases should have the examination repeated and further dye given on the evening of the day of X-ray examination, or perhaps even subsequently. But the error is very small and, while ideal, it may not be justifiable economically. Because of this occasional failure of the gall-bladder to fill with the oral method it is sometimes advocated that the intravenous method should be employed.

*Intravenous technique versus oral technique.*—Kirklin, who has for a long time advocated the oral method, has had experience of both methods. He decided that the method of choice was the oral one for greater ease of examination and equal reliability. One could also argue that if the intravenous method gave a better shadow, as it is claimed to do, then it would appear from the evidence already presented that it might even tend to increase the inaccuracy of the reports because a greater number of cases would fall into the normally functioning group. Largely as the result of Kirklin's work and the high accuracy obtained at the Mayo Clinic, the oral method is now most frequently employed.

#### *Operative Findings in the Poorly Functioning and Non-functioning Cases*

It is usually thought that the failure of the gall-bladder to concentrate the dye satisfactorily must be due to disease within the gall-bladder. While this is in the

main true, disease in other sites may be also responsible. The actual operative findings have been set out in the following two tables :—

*Interpretation of the Term "Poorly Functioning" Gall-bladder Without Stones in Terms of Pathology.*

Cholecystitis and gall-stones	..	22
Metastases in liver	..	1
Carcinoma of hepatic flexure secondarily involving the gall-bladder	..	1
Total	..	24

*Interpretation of the Term "Non-functioning" Gall-bladder Without Stones in Terms of Pathology.*

Cholecystitis and gall-stones	..	109
Carcinoma of the gall-bladder	..	2
Primary carcinoma of liver and metastases in the liver	..	4
Cirrhosis of the liver	..	1
Carcinoma of the head of the pancreas	..	5
Acute pancreatitis with dilatation of the gall-bladder	..	1
Carcinoma of the common bile-duct	..	1
Normal	..	4
Total	..	127

It will be noted (if the errors are excluded) that the findings can be classified under four main headings. To the above groups have been added a series of 157 cases personally investigated at the Mayo Clinic.

*Analysis of "poorly functioning" and "non-functioning" gall-bladders without stones (Edinburgh Cases Plus a Series of 157 from the Mayo Clinic).*

		<i>Per cent.</i>
Disease of the liver	6	1.9
Disease of the gall-bladder or gall-bladder packed with stones	280	90.9
Obstruction of common bile-duct	13	4.2
Extrinsic causes secondarily affecting the gall-bladder	2	0.7
Gall-bladder normal	7	2.3
Total	308	100

It will be noted that in 6.8% of cases the cause is elsewhere than in the gall-bladder. Obstruction of the common bile-duct accounts for 4.2% of the cases. While the liver may continue to excrete sufficient dye when extensively diseased, a poor or absent shadow may result in certain cases. This is especially likely to occur if the disease affects the main ducts within the liver as in one case in the Edinburgh series, where a large primary carcinoma of the liver was found near the gall-bladder. Under the heading of extrinsic causes secondarily affecting the gall-bladder I have included a large carcinoma of the hepatic flexure, which was invading an otherwise healthy gall-bladder, and one case of a fistula between the gall-bladder and the colon. In 90.9% of cases the failure to obtain a good shadow was due to cholecystitis or to gall-stones so tightly packed in the gall-bladder that it was either unable to cast a shadow at all, or to produce only a very faint shadow. In a few cases carcinoma of the gall-bladder was present.

The frequency of malignant disease in the Edinburgh series is perhaps higher than that usually found. This is explained by the method of obtaining the operative

and autopsy findings. In addition to depending on the house surgeons to make returns of the operative findings, a separate and more complete investigation is made of all cases of malignant disease attending the Royal Infirmary.

*The effect of the presence of gastric or duodenal ulcers.*—In the Edinburgh series four duodenal ulcers were found in the normally functioning group. Three duodenal ulcers were present in the combined poorly and non-functioning groups. In the four errors in the non-functioning group there were no peptic ulcers present. The series, however, is much too small to be conclusive and I will, therefore, make use of some Mayo Clinic cases which I personally investigated. The findings in the larger series are as follows :—

In a group of 350 normally functioning cases duodenal ulcers were found in 60 or 17% of the total.

In a combined series of 311 poorly and non-functioning cases duodenal ulcers were found in six cases. Of these six, four had disease of the gall-bladder and two were normal. If the duodenal ulcers were responsible for the failure to obtain a good shadow in these two cases then they give rise to only 0.6% of errors as a maximum.

These figures show clearly that the presence of a duodenal ulcer at least did not appear to influence the density of the gall-bladder shadow. In cases, however, of organic stenosis resulting from duodenal ulcer, the rate of emptying of the stomach will be delayed and there will be interference with the absorption of the dye from the bowel. In these circumstances the gall-bladder shadow will be faint, but the presence of the dye in the stomach may be noted on the film and the correct interpretation of the faint shadow made.

Gastric ulcers and carcinoma of the stomach may give rise to a similar set of conditions and may then interfere with the concentration of the dye.

*The significance of a primary shadow.*—In a small percentage of cases the gall-bladder may cast a shadow of sufficient density to render it visible on the plain film. When this takes place the visualization is due to a high calcium content in a diseased gall-bladder and such a high calcium content is found only if the cystic duct is blocked. Similarly calcification of the gall-bladder wall is found only if the cystic duct is blocked. Opacal is therefore unnecessary, for no dye can enter the gall-bladder.

Statements that the normal gall-bladder may be visible on a plain film are usually due to confusion with the shadow of the filled duodenal cap or to the liver shadow overlapping the outer margin of the kidney.

*The diagnosis of adhesions from deformity of the gall-bladder shadow.*—Kirklin, after investigating a large series of cases, found that deformity of the gall-bladder shadow may be unaccompanied by adhesions. Conversely, a gall-bladder buried in adhesions may be associated with a shadow of normal outline.

Cholecystography is therefore an unreliable method of determining the presence or absence of adhesions.

*The purpose of the fat meal.*—The fat meal is not given to determine the rate of emptying of the gall-bladder. It has been pointed out in another paper that this is almost certainly of no significance. Similarly it is not given to see if the gall-bladder will empty, for any gall-bladder which fills will certainly empty. If an obstruction exists the gall-bladder fails to fill at all.

The fat meal is given to contract the gall-bladder and so render visible small stones and tumours.

*Tumours of the gall-bladder.*—In the two cases of carcinoma of the gall-bladder in the Edinburgh series, the gall-bladder was of the non-functioning type. This is the usual finding.

Only one case of simple tumour of the gall-bladder has been reported in Edinburgh and the case did not come to operation. Simple tumours of the gall-bladder were

first described by Kirklin, and I would refer those interested to his published work on this subject.

It is hoped that if the suggestions put forward in this paper are adopted there will be a better understanding between radiologists and clinicians when cholecystographic examinations require to be made. Figures have been presented showing the findings obtained when such methods are put into operation.

In conclusion, I wish to express my thanks to Dr. J. P. McGibbon of the Department for his valuable assistance in collecting the operative findings.

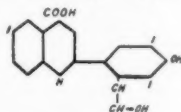
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**Dr. H. W. Gillespie** said that it was fifteen years since Graham and Cole introduced into radiology the use of the sodiumtetraiodophenolphthalein. However, this phenolphthalein group represented only one among numerous other substances which are mainly excreted by the liver, e.g. Azo dyes. The solubility in water of these drugs played an important role, and the less easily one of the drugs of the aromatic group was dissolved the more likely it was to be excreted by the liver, and the more soluble it was, the easier it was excreted by the kidneys. One would imagine that by taking any of these drugs and iodizing it, one could obtain a new contrast dye, but by the addition of iodine a drug might easily become toxic. A further difficulty was to attach the iodine firmly to the molecule, so that it was not split off on its way from the intestines to the liver.

Dr. Gillespie described his experiences of cholecystography with a dye of the quinoline group which has been mentioned by Pribram in 1936. This was a yellow powder, with no taste, not easily soluble, and not unduly toxic, as, according to Pribram, a rabbit tolerates 2 grm. of the substance per kilogramme of its weight without any disturbance. Chemical formula:—

2—(3'-5' Diiodo-2' hydroxyethyl-4' hydroxyphenyl) -6-iodo-4 quinolinic acid.



The results in a series of ten patients were mentioned, where the gall-bladder shadow produced by  $2\frac{1}{2}$  grm. of the new dye equalled in density the shadow usually produced by 4 grm. of sodiumtetraiodophenolphthalein. A series of slides was shown comparing the shadow density in the same individual after the oral administration of 4 grm. of sodiumtetraiodophenolphthalein, and at a later date after the oral ingestion of  $2\frac{1}{2}$  grm. of the new dye. Out of six cases, a denser shadow was produced by the new dye in three cases, in one case the density was equal, and in one case the sodiumtetraiodophenolphthalein produced a better shadow. In one case the gall-bladder was not visualized after sodiumtetraiodophenolphthalein, but showed up after the administration of the new dye. A feeling of nausea was experienced in two of the six cases after the ingestion of the new dye.

The advantage of the new dye is that it produces a denser and possibly more reliable shadow, the disadvantage is its close chemical affinity to atophan or cinquophen. However, in comparing this drug with cinquophen it is pointed out that for cholecystographic purposes it is usually only used once.

On account of the small quantity of dye available and the limited number of investigations, no definite opinion is given on its usefulness.

[January 19, 1940]

## DISCUSSION ON THE TREATMENT OF METASTASES

**Dr. F. M. Allchin:** Ewing says that the formation of secondary tumours is a cardinal property of malignant tumour growth. It is regarded by those who treat malignant disease as almost inevitable, that every case of cancer will at some stage or other of its history develop metastases. The date at which these may appear is subject to enormous variation, and some types are more prone to metastasize than others. The radiosensitivity of metastases is certainly not greater than that of the primary growths from which they originated, and in very many instances is unfortunately much less.

Every patient with secondary cancer should be the subject of a general consultation including of course the radiotherapist, before treatment is undertaken, or the dreaded word given that nothing can be done. With increasing experience it is often found that cases appearing to be almost hopeless respond well to treatment, whilst others that seem generally in good condition do very badly. It must, however, be recognized that it is necessary to distinguish the type of case in which the tissues, through long-standing disease, the state of the blood, or the general metabolic activity of the body, render it likely that the response to radiation may be negligible or useless.

General principles of treatment: In order that the best results may be obtained all forms of radiation should be available. X-rays of supervoltage, of 200 K.V. and low voltage (60 K.V.) may all be used as well as radium teletherapy, radium by superficial plaques, and radium interstitially. There are times when two or even more of these forms may have to be employed to bring about the desired result. Details of dosage depend on many factors, but as a general rule the total 'r' figures will have to equal those necessary in attacking the corresponding primary and in many instances to exceed these. In some cases, notably the bony deposits from breast cancer, satisfactory results can be obtained with much smaller doses. If deposits are solitary or single there should always be borne in mind the possibilities of surgery or diathermy. In all cases of generalized disease the state of the blood must be investigated before treatment is begun and counts should be repeated at regular intervals during the course. All necessary measures must be taken to counteract anæmia even to the use of transfusions.

Rest in bed is essential in many cases, especially those with bony deposits, and should be continued even after the cessation of treatment. Splinting and the use of supports may be necessary in these cases, and the addition of calcium therapy is often beneficial. In every case the general condition of the patient must be rigidly attended to and no detail omitted which it is thought may assist in overcoming the disease and adding to the comfort of the patient. In all cases the relatives of the patient should be told the nature of the trouble. The aims of the treatment and especially its limitations should be clearly pointed out in order that false hopes be not raised.

In the future it is hoped that additional aids may be found to supplement the effects of radiation. Many such have been used in the past but without conspicuous success.

At the present time radiation offers the only reasonable chance of alleviation in widespread cases. Apart from those cases where it is possible to use surgery or diathermy, radiation is the method of choice. In a very large number arrest of the disease is brought about and pain is relieved, this latter being more lasting than that produced by sedative drugs and without the disadvantages.

**Dr. Margaret C. Tod:** The principles of radiotherapy are fundamentally the same whether the lesion be primary or metastatic. The first decision is always whether treatment can be expected to cure or only to palliate, but with this difference, that once metastases are present the proportion of cases for which cure is possible falls very low. The curability of metastases by any method, including surgery, depends greatly on the route by which the neoplastic cells have reached the site of secondary deposit. When the spread is by the lymphatics the next site likely to be involved can be foretold with some certainty. Treatment can then be planned to deal with the spread before it becomes evident, the outstanding example being the radical operation for cancer of the breast carried out before there is clinical evidence that lymph nodes are involved. X-ray fields are also planned with prophylaxis in view and can go further than surgery in the treatment of cancer of the breast by including the supraclavicular lymph nodes. It must be made clear that this treatment is not prophylactic in the sense that it prevents future involvement; rather it attempts to cure small secondaries which may already have reached the lymph nodes without producing any change which can be detected clinically. When radiation follows surgery the aim is again to destroy small groups of neoplastic cells left behind. Such treatment is curative, and it is only when spread by the lymphatics extends to regions where curative dosage is no longer possible that treatment of this form of metastases must become palliative. Some neoplasms produce metastases by invading the blood-stream, when secondary deposits may occur in any organ, and they are usually multiple; rarely they may be single. It is impossible to attempt to forestall the appearance of blood-borne metastases either before they appear or after the detection of a solitary deposit, so that their treatment must in general be palliative, spread being followed by therapy, a method later described as a "chasing" technique.

In spite of the influence of method of spread on curability it is less important in the decision regarding method of treatment than is the radiosensitivity of the particular neoplasm to be treated. For purposes of this discussion radiosensitivity may be defined as that property of the neoplastic cell which determines the dose of radiation required to produce complete and permanent regression of a tumour within the treated zone.

Metastases from two groups of tumour are susceptible of curative therapy, highly radiosensitive tumours which call for treatment of large volumes at low dosage, and tumours of limited sensitivity, a group which includes the familiar squamous carcinoma and most of the carcinomas of the breast. These must be treated with higher doses and therefore in small volumes. The treatment of metastases from resistant tumours such as the sarcomas of adult connective tissue is never curative.

The main problem which has to be decided before treatment is undertaken is the probable sensitivity of the tumour. If a pathological diagnosis or a firm clinical diagnosis has been made dosage can be decided on knowledge of the nature of the neoplasm, but if pathology is indeterminate or biopsy impossible, it may be necessary to test the sensitivity of the obvious lesion either by giving a small dose to the whole lesion or a rather larger dose to a small part of the tumour, and watching the effect.

*Methods of curative treatment.*—The methods which may be considered as curative are:—

- (1) Regional therapy for tumours of high sensitivity.
- (2) Localized therapy for tumours of limited sensitivity.

If very large fields must be used, general tolerance becomes the limiting factor and doses must necessarily be low. Opinion is not yet unanimous regarding the radiosensitivity of many tumours, but the following list includes those which are accepted by the Holt Radium Institute as probably highly sensitive and treated by regional therapy.

(i) *Embryonal origin.*

Seminoma testis.

True embryonal tumour of ovary.

(ii) *Primitive tumours of unknown origin.*

Wilm's tumour of kidney.

Some tumours of thyroid glands.

Some tumours of salivary glands.

Some tumours of nasopharynx and nasal accessory sinuses.

(iii) *Reticulo-endothelial origin.*

Leukæmia.

Hodgkin's disease (lymphadenoma).

Lymphosarcoma (reticulosarcoma).

Thymoma.

Ewing's tumour.

Endothelioma (of vascular origin).

Miscellaneous reticulo-endothelial diseases.

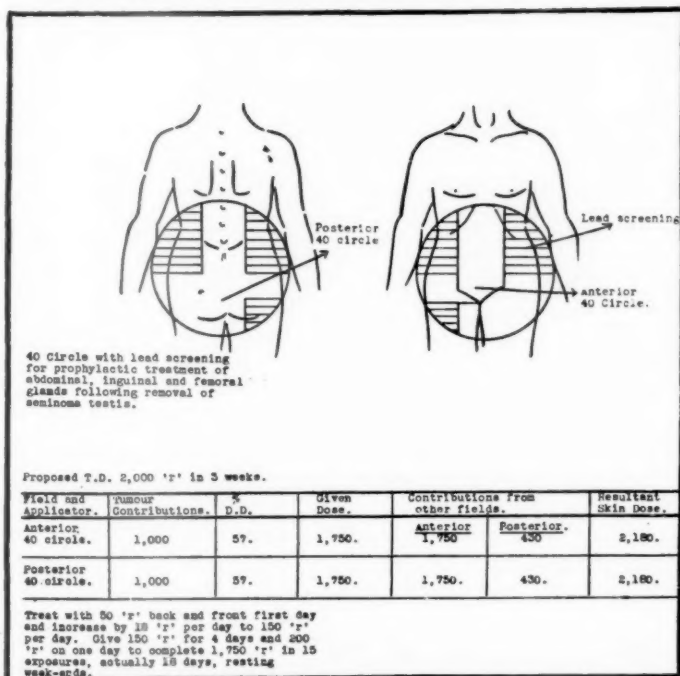


FIG. 1.

*Methods of treatment.*—Regional therapy aims at delivering to the whole region likely to be involved doses of radiation which should prove lethal to the tumour without exceeding general tolerance. X-radiation is always the method of choice in the treatment of the highly sensitive tumours because of the tendency to early and widespread metastases or the multiple foci of neoplasia.

In discussing X-ray therapy it is taken for granted that except where contact therapy is specified, filtered X-radiation generated at 200 K.V. or over is employed, and that the tumour dose is that measured or calculated at the estimated depth of the tumour, skin dose being measured with back scatter. Gamma ray dosage is also expressed in roentgens.

An important example of a typical regional arrangement is applied to the treatment of the very sensitive seminoma testis, and this is shown diagrammatically.



FIG. 2.

The dose aimed at is 2,000 'r' at the mid-point between two large fields, anterior and posterior.

The treatment if abdominal secondaries are already present is exactly the same as the prophylactic treatment. In the treatment of such large areas general tolerance usually becomes exhausted, and it is necessary to watch the blood-count very carefully as this is the best index of general tolerance. It has been shown by David Goodfellow that the total lymphocyte count is the best indication of the effect on hæmopoiesis. The total white count also falls and finally the red count, but these follow the fall in lymphocytes. Serious consequences can therefore be avoided if radiation is stopped

when the lymphocytes reach a point at which recovery is still known to be possible. We have found that an absolute count of 300 lymphocytes may be regarded as safe. Patients can recover when the lymphocyte count has dropped below 200, but there is a tendency for the count to continue to fall for some days after radiation stops, and the figure of 300 leaves a margin of safety.

For the abdomen, treatment can begin with 50 'r' back and front increasing by 10 'r' daily to 150 'r' per day. The thorax is less tolerant and it may be necessary to begin with 30/50 'r' per day increasing by 5 'r' daily to 120 'r' per day according to the systemic reaction. The low tolerance of the thorax is a definite finding in our clinic, though not in agreement with some observers (fig. 2).

In general the problem of treating the highly sensitive metastases is not one of obtaining a dose, but of covering enough ground to deal with all the foci without exceeding general tolerance.

*Localized therapy for metastases from tumours of limited sensitivity.*—If regional therapy is a question of covering ground, the therapy of tumours of limited sensitivity is essentially a question of dose. The common accessible tumours nearly all belong to this class, and experience has proved that when an attempt is made to cure squamous or columnar-cell carcinoma a certain minimum dose must be attained if there is to be any hope of success. The dose required is such that the volume of tissue which can be treated is strictly limited by local tolerance. It must be clearly understood that when radiation is discussed as a treatment for secondary deposits from tumours of limited sensitivity, operable metastases in lymph nodes are excluded. Operation, if possible, must be given first place. This dogmatism may seem strange when it is remembered that for primary cancer of the mouth radiation is our method of choice, but whether the secondary deposits are more resistant, as some observers maintain, or whether they have the same sensitivity, surgery deals with the whole cervical chain and therefore treats a volume of tissue which could not be raised to the dose which can be obtained by implant or small field X-ray therapy of the curable primary.

If curative X-ray therapy is attempted, full biological dosage is needed even if much trouble must be taken to direct the beam and to chart the depth doses. We have found doses of 6,000 'r' in five weeks, or 4,000 'r' in four days to be suitable dose levels for tumours of limited sensitivity.

*Palliative therapy of metastases.*—Four techniques for palliative therapy merit description.

(i) Localized therapy for single deposits of sensitive neoplasms when more foci are known to be present.

(ii) A growth restraint technique for resistant tumours.

(iii) A "chasing" technique which treats individually a series of secondaries as they appear.

(iv) Symptomatic palliative therapy.

Before discussing the methods separately, the principle of palliation is recalled. The obvious requirement that palliative treatment should palliate is sometimes forgotten, and treatment is given to cases impossible of cure which produces severe local or general reactions. This is unjustifiable and palliative treatment must either be kept at dose levels which do not produce serious reactions or else the reaction produced must be strictly limited to a small area where it is desired to heal an ulcerated surface or relieve tension which is causing pain. When considering palliative therapy the probable length of life should be assessed. Treatment likely to occupy most of the remaining time can seldom be desirable.

**Dr. N. S. Finzi:** Having had a large number of such cases to treat I have experience going a long way back, and therefore would like to say a few words as to curability. I have one case who remained well for thirty years and others of twenty-five years, sixteen years, and several for ten years. I think in the circumstances we can say that some of these cases are curable. Of course in the early ones it was only an accident that they recovered, because, though I did not know it at the time, I know now that their treatment was very bad.

The whole subject of the treatment of radio-sensitive metastases has altered in the last few years. About 1926 I treated a patient with multiple glandular metastases for what was then called lymphosarcoma, with a thoracic bath, and he remained well for some years. I also treated cases with widespread abdominal radiation. I never had the courage, however, to treat the whole of the thorax and abdomen together, and it remained for my colleague, Dr. Levitt, to do this. He has now a number of cases which have remained well for five years with such treatment and he will shortly publish a summary of his results.

## Clinical Section

President—DUNCAN FITZWILLIAMS, C.M.G., F.R.C.S.

[March 7, 1940]

### Xanthoma Diabeticorum with Lipodystrophia.—R. D. LAWRENCE, M.D.

This was a case of *diabetes mellitus* accompanied by xanthomatosis, persistent lipæmia, and hypercholesterolaemia, extreme hepatosplenomegaly, lipodystrophy, enlargement and fibrosis of lymphatic and parotid glands (by biopsy) and high metabolic rate.

A woman of 26 developed diabetes and showed extreme lipæmia (6%) and eruptive xanthomatosis in 1936. In spite of treatment with adequate carbohydrate and insulin, lipæmia persisted (2%) and hepatosplenomegaly developed. High carbohydrate fat-free diets made no fundamental difference to the condition. Later lipodystrophy of the face, shoulders, and hands developed, and enlargement of parotids and the general lymphatic glands became obvious. The basal metabolic rate is persistently between +50 and +75%. The case does not fit into any hitherto described syndrome, but resembles a case briefly described by Ziegler (*Brain*, 1928, 51, 149). Splenic and hepatic punctures have failed to throw light on the fundamental pathology.

Dr. F. PARKES WEBER said that this case was unique, because of the great variety of the rare features which were associated with the diabetes mellitus. One knew that diabetes mellitus might be associated with one or more of the following features: (1) lipæmia; (2) retinal lipæmia; (3) hypercholesterolaemia; (4) eruptive cutaneous xanthomatosis; (5) *chronic enlargement of spleen and liver*, a very rare condition, apparently associated with permeation of the affected viscera by cholesterol-containing "large clear cells". As to the *raised basal metabolism*, an investigation would be required to ascertain how often this was present in diabetics. As to the *lipodystrophia superior progressiva* (not necessarily progressive) he (Dr. Weber) had never heard of its association with diabetes mellitus and was most interested to know that such an association had been observed in the Mayo Clinic. He (Dr. Weber) had likewise never heard of the association of diabetes with an *atrophic symmetrical sclerosis of the parotid salivary glands*, as in the present case. The "biopsy" on one of the parotid glands seemed to show replacement of salivary gland parenchyma by lymphocytes and true lymph-follicles—just as in the condition known as "lymphadenoid goitre" the true thyroid gland parenchyma became gradually replaced by lymphocytes and lymph-follicles. The condition of the salivary glands in this patient should be compared with the changes in "Sjögren's syndrome" (see F. Parkes Weber and A. Schlüter, *Deut. Arch. klin. Med.*, 1937, 180, 333).

### Fractured Lumbar Vertebra.—J. A. SEYMOUR-JONES, M.B.

A. B., female, aged 10 years.

*History*.—6.2.40: Admitted to the Hampstead General Hospital under the care of Mr. Cameron MacLeod with the complaint of pain in the back.

She fell down some stairs in August 1939, but was able to get up unaided, and no ill-effects were noted.

December 1939, shortly before Christmas, she again fell down a flight of stone stairs in the house to which she had been evacuated. She got up without assistance and since then has been attending school, but complained of aching pain in the back, which was worse at night.

A third fall occurred a few days prior to admission. The pain became worse and her mother took her to the Hospital Out-patient Department.

*Previous illnesses*.—Chicken-pox, measles, mumps. No family history of tuberculosis.

*On examination*.—She is a well-nourished, intelligent child. In the dorsi-lumbar region of the vertebral column there is a well-marked sharply angulated kyphus.

There is no local tenderness, minimal muscle spasm or wasting, no pain on percussing spinous processes or crown of head. Some limitation of spinal movements in all directions. No alteration of tendon reflexes and no sensory disturbances noted.

*X-ray appearances* (Dr. Rohan Williams): (1) "Gross destruction with compression is present in the bodies of D.V. 12 and L.V. 1 which have 'mushed' together causing angular kyphus." (2) A minor compression injury is also present in body of L.V. 4.

Lungs: No lesion seen.

Selected skeletal studies: Bone growth and formation quite normal. No suggestion of any form of fragilitas ossium to account for vertebral injuries.

*Investigations*.—February 16: Hb. 97%; W.B.C. 6,800 per c.mm. *Differential count*: Polymorph-neutrophils 64%, 4,352 per c.mm.; eosinophils 3%, 204 per c.mm.; lymphocytes 25%, 1,700 per c.mm.; monocytes 8%, 544 per c.mm.

Stained film: Appearance normal.

Blood sedimentation rate: 4 mm. at one hour.

February 17: Serum calcium 10.8 mgm. per 100 c.c. Plasma inorganic phosphorus 2 mgm. per 100 c.c.

Mantoux test positive.

Vollmer patch test (Lederle) positive.

Since admission the child has been kept in bed, and no weight-bearing permitted, but no form of extension or plaster has been applied.

#### **Idiopathic Lymphœdema.—E. HERZBERG, M.D.**

Mrs. F., aged 27.

*History*.—Nine years ago had frequent attacks of tonsillitis. One lasted for over six weeks, in the course of which swelling developed in the middle phalanx of the middle finger of the right hand. This swelling spread to the other fingers, though it never extended beyond the metacarpus. From that time until July 1938 she was perfectly healthy, except for a difficult confinement in 1936, after which she had some obscure febrile illness. July 1938, painless swelling of the right hand, gradually extending to the forearm; was treated for rheumatism. Then seen by Mr. John Simons of Tunbridge Wells, who advised removal of her tonsils. After the operation, November 1938, the swelling went down for about two weeks, then gradually increased again and remained stationary until June 1939, when first seen by me. Since that time there has been gradual diminution of swelling and the circumference of the arm has decreased by about an inch.

*Investigations*.—Blood-count: Hb. 80%; erythrocytes 4,820,000; C.I. 0.83; mean cell diameter  $7.3\mu$ ; leucocytes 4,600. *Differential count*: Basophils 0.5%; segmented 45%; lymphocytes 47.5%; monocytes 6.5%; plasma cells 0.5%.

Urine: Sugar negative. Albumin negative.

Wassermann reaction negative.

X-ray of chest negative.

Dr. PARKES WEBER agreed that the case was one of idiopathic "lymphœdema", a condition which he had usually termed the *Nonne-Milroy-Meige type* of œdema of extremities, which was often more or less familial and hereditary in incidence, but very rarely was localized to an upper extremity, as in the present case.

#### **Scleroderma with Involvement of Muscles.—R. S. BRUCE PEARSON, D.M.**

T. F., male, aged 34.

*History*.—During the summer of 1938 patient experienced pain in the terminal part of the first finger of each hand, the finger-tips turned black and a small portion of each eventually separated. From this time he noticed that his hands and feet were frequently cold and blue.

In June 1939 he had a right cervical sympathectomy carried out. This has led to no improvement. About this time he noticed that his hands, shoulders, and knees

were becoming stiff and this has increased since. He used previously to have a high colour and recently this has disappeared and he has lost 3 st. in weight.

*On examination.*—His face is drawn and expressionless. The skin over his hands and forearms is contracted and adherent and prevents him from closing his hands. There is no swelling of the joints but on moving wrists, knees, or elbows, a creaking noise of muscular origin is audible and movement is limited. His hands and feet are usually cold to the touch, but on occasion are quite warm. No difference in temperature can be detected between the right and left arm but he has a Horner's syndrome on the right side.

**Chronic Purpura following Rheumatic Fever.**—ELI DAVIS, M.D.

(By permission of Dr. D. S. Sandiland, Medical Superintendent, St. Stephen's L.C.C. Hospital.)

Female, aged 33. Little is known of her family, but her father died of pulmonary tuberculosis.

*History.*—Had rheumatic fever when 7 years old and four acute attacks since. She had her third attack at 19 and was in bed eight months. On getting up, ecchymoses appeared on her thighs and legs. They recurred every few weeks, often preceded by pains, aching and stiffness in the lower limbs. In the last few years, ecchymoses appeared much more frequently. I first saw her in September 1938 in St. Stephen's L.C.C. Hospital, and from then on I have a continuous daily record of her ecchymoses. She has had an average of about 15 ecchymoses per week (usually painless), from  $\frac{1}{4}$  in. to 4 in. in diameter (average  $\frac{3}{4}$  in.) mostly on the legs and thighs but occasionally on the arms. Crops of petechiae were sometimes seen. On getting up after a number of days in bed, purpura becomes particularly evident.

*On examination.*—Mitral stenosis and aortic regurgitation are present. There is no cardiac failure. The patient is up several hours a day. Blood investigations are normal. The capillary resistance test is variable, but has often been positive. Vitamins C and P did not influence the purpura. There is no other unusual bleeding or bruising.

*Comment.*—A. F. Coburn in 1931 ("Factor of Infection in the Rheumatic State", Baltimore) described a prolonged follow-up of 162 rheumatic patients, in 11 of whom purpura was observed at various times. But he did not mention persistent purpura following acute rheumatic fever.

Dr. BRUCE PEARSON thought that it would be unwise to assume that there was any causal relationship between the purpura and rheumatic fever. Although the patient had a valvular heart lesion indicating past rheumatic disease, there was no evidence of any activity of the rheumatic agent at present, whereas the purpuric state continued.

**Chronic Purpura, with Orthostatic Factor, following Trauma.**—ELI DAVIS, M.D. (By permission of Dr. D. S. Sandiland, Medical Superintendent, St. Stephen's L.C.C. Hospital.)

Female, aged 26. Cook.

*History.*—27.1.39: She fell downstairs and bruising of the left knee was seen next morning. Examination of the joint by X-ray and under anaesthesia at St. James's L.C.C. Hospital showed no internal derangement. She improved.

June 1939: Readmitted for a fresh haematoma around this knee. More ecchymoses appeared while in hospital, especially on getting up.

1.11.39: She entered St. Stephen's L.C.C. Hospital as painful ecchymoses continued to appear in the absence of any injuries. Mr. H. K. Vernon kindly referred her to me. Several raised tender ecchymoses were seen around the left knee and upper leg. The leg was cedematous and she limped. In hospital, numerous purpuric areas appeared, and although they often arose when she was strictly confined to bed, their number and size always increased markedly when she got up. For example, after prolonged rest in bed most lesions had faded, but on sitting for half an hour with her feet touching the floor, four new ecchymoses appeared. The purpura was

at first localized to the knee, but later the leg and then the thigh and hip became extensively affected. Large doses of vitamins C, P, and B did not help. Apart from menorrhagia in the last two years, there was no other abnormal bruising or bleeding in the patient or her family.

*Investigations.*—Blood-count: R.B.C. 4,640,000; Hb. 78%; W.B.C. 7,900. Differential count normal. Platelets 270,000. Bleeding and coagulation time normal. Blood Wassermann reaction and Kahn test negative. Blood sedimentation rate: 18 mm. in one hour. Capillary resistance test negative.

Temperature and pulse normal throughout.

*Comment.*—It is of note that purpura appeared in this patient after sitting up for half an hour, and that the ecchymoses were confined to the injured limb.

Gruss (1919) and Hirsch (1921) have published papers on "Traumatic Purpura"; Weir Mitchell (1869) described patients in whom severe neuralgic pain was followed by purpura, and Castex (1924) claimed a neurological aetiology for the condition.

The patient under discussion has severe pain in her left lower limb, but examinations of the nervous system and cerebrospinal fluid are negative apart from moderately depressed appreciation of all forms of sensation over the ecchymoses.

Deléarde and Hallez (1912) described a girl who was frightened when a carriage in which she was riding at a fair became derailed; there was no physical injury; repeated attacks of purpura, chiefly over the lower limbs, occurred after fatigue or emotion, and the limbs were noted to be cold before the purpura appeared; vasomotor influences, as suggested by the authors, do not adequately explain these manifestations. In the patient under discussion the left foot is persistently cold, the purpura and the pain are becoming increasingly severe, and there is progressive difficulty in walking even short distances.

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*Discussion.*—Dr. ERIC WITTKOWER: The accident which preceded the onset of purpura in Dr. Davis's case occurred during a period of severe emotional strain (discovery of unfaithfulness of fiancé, broken-off engagement). Subcutaneous hæmorrhages, if psychologically conditioned, may be artefacts or stigmata. Careful psychological examination does not suggest either of these mechanisms in this case.

Dr. J. D. ROLLESTON said that he had seen several examples of extensive purpura as a sequel of the acute exanthemata. The issue in such cases was usually fatal. He had seen, however, a very severe case of purpura reported by L. J. M. Laurent following a mild attack of measles in an infant, aged 8 months, who had recovered after intramuscular injection of streptococcal antitoxin (scarlatinal) followed two days later, as there was no improvement, by intramuscular injection of the fresh uncitrated blood from the mother. He suggested that similar treatment might be tried in the present case. He did not think the lesions were self-inflicted.

*Reference.*—LAURENT, L. J. M. (1933), *Brit. J. Child. Dis.*, **30**, 104.

*POSTSCRIPT.*—It was suggested at the meeting that the patient's left lower limb be encased in plaster. This was done by Mr. H. K. Vernon, and it was removed after twenty days. There were four new purpuric areas over the leg and thigh. The limb has improved, however, and the patient walks better. Purpura has also appeared extensively over the left upper limb and slightly over the right lower limb. The left hand and forearm are colder than the right.—E. D.

**Acrocyanosis, Hyperpiesis, and Multiple Arthritis.**—G. J. DIXON, M.R.C.P.

A. B., male, aged 62. Salesman and manager.

*History.*—Ten years ago suffered from attacks of pain, swelling and limitation of movement of elbows, knees, wrists, and hands; this came on gradually, but became

sufficiently severe for him to spend two or three days in bed ; gradual recovery over two or three weeks.

He has had several similar milder attacks since. Since the first attack his hands readily become very cold and excessively blue. He has been getting increasingly short of breath on exertion during the last three years. About two years ago he developed some pale, reddish-blue spots over his face—these have been present ever since.

On Saturday, December 30, he awakened with a start, possibly caused by the ringing of a bell, and found that the vision of the left eye was misty ; this phenomenon decided him to seek medical advice.

*On examination.*—A thin man, with reddish-blue nose and multiple telangiectases over face. When first seen the hands were excessively cold and the terminal portions of the phalanges were swollen and clubbed and of a deep blue-grey colour.

At other times there was less swelling of the digits, and though the ends were clubbed the skin was loose over these clubbed ends ; the nails were freely movable over the terminal phalanges ; at such times the discoloration was much less marked.

There was limitation of extension of both elbows and limitation of extension and flexion of both knees accompanied by grating. Very well-marked hallux valgus.

*Investigations.*—In January the right fundus showed some arteriosclerosis, the left fundus showed large dilated veins with numerous flame-shaped hæmorrhages along their course, especially in the left lower quadrant. There were a few hæmorrhages along the course of the retinal arteries. There was some blurring of the disc margins.

The heart was enlarged and when first seen the patient was noticeably short of breath, with a pulse-rate of 95. His blood-pressure was 210/100. There was well-marked Mönckeberg's sclerosis of the peripheral vessels.

X-ray confirmed enlargement of heart and showed pulmonary congestion. Elbow-joint : showed presence of loose body. Hands : showed disappearance of distal portion of terminal phalanges.

Dr. PARKES WEBER thought that this case belonged to the class of generalized symmetrical scleroderma. The association of acrocyanosis in the thickened hands was typical—as also was the association with multiple cutaneous telangiectases in the slightly sclerodermatous face. Disappearance of the distal portion of the terminal phalangeal bones occurred in some such cases, even when the bone-atrophy could not merely be due to pressure of sclerodermatous tissue.

**Congenital Megacolon treated by High Spinal Anaesthesia.**—G. C. DORLING, F.R.C.S. (*By permission of the Medical Superintendent, Lewisham Hospital, and of the Chief Medical Officer, L.C.C.*)

J. M., female, aged 3½ years, second child.

*History.*—Constipated since birth. Bowels open only once a week, after enema.

*On examination.*—31.1.40 : Admitted to Lewisham Hospital. Dry skin, sallow complexion. Lethargic. Undersized. Weight 30 lb. Bowels had not been opened for seven days. Abdomen was slightly distended and the rectum was loaded with hard faeces.

Straight X-ray showed gaseous distension of the colon. Barium enema 2.2.40. Distal colon grossly distended as far as middle of transverse colon. Scanty haustration distal to this point. Hirschsprung's disease.

During ten days' observation in hospital, bowels open once.

On 8.2.40 a spinal anaesthetic was given by Dr. Wolfson producing a sensory block up to D.6. 4.5 c.c. of 1 : 1,500 percaine was used by the Howard Jones technique.

Since then her bowels have acted regularly and she has not required a single enema. After the first week she has had two actions daily. Liquid paraffin was administered during the first fortnight.

Barium enema 11.2.40 : The colon is about the same size, but haustration is better. Only about a quarter the quantity of barium is present twenty-four hours later.

**Cyst of the Left Iliac Bone.**—ZACHARY COPE, M.S.

The patient is a well-developed young woman of 19 years who has complained of pain in the region of the *right* hip, buttock, and outer aspect of the right thigh for four months.

*Past history.*—Some years ago she was treated for mild scoliosis.

*On examination.*—Both hips were found to be normal but there was prominence of the right posterior sacro-iliac region and some asymmetry of the pelvis. No shortening of either limb. X-ray examination showed obliquity of the pelvis with deficient development of the left side of the pelvic girdle.

A large cystic space was shown in the left ilium close to the left sacro-iliac joint.

*Diagnosis.*—Simple cyst or possibly osteoclastoma.

*Operation* (22.2.40).—Cyst exposed by crescentic incision. Thin-walled posteriorly. Contained blood-stained fluid and presented a definite lining which was scraped away. There was a smaller loculus which opened out of the main cyst. Interior wiped out with solution of zinc chloride.

Immediately after recovering from the anæsthetic the patient said that she had completely lost all the pain on the right side and this freedom from pain has been maintained, though the cyst was on the *left* side.

Microscopical examination showed the lining to consist of amorphous debris and organized blood-clot. There was no evidence of osteoclastoma.

**Spasm of the Œsophagus.**—DUNCAN FITZWILLIAMS, F.R.C.S.

Miss O'M., aged 52.

*History.*—She had her right breast removed in Cork for carcinoma nearly three years ago. She remained quite well till about six months ago, when she began to have some difficulty in swallowing. A little later she noticed a mass coming in the right side of her neck just above the clavicle. She can now swallow only fluids and soft solids.

*On examination* (6.2.40).—She was thin and had been losing weight rapidly. There was a mass of hard glands above the right clavicle, slight stridor, but nothing else could be made out.

It was thought probable that she had some mediastinal metastasis pressing on her œsophagus giving rise to the symptoms. An X-ray was taken and the mediastinum was found quite clear of all suspicion of metastasis. A narrowing of the œsophagus for 6 to 8 in. was seen, quite smooth, which appeared to be a simple spasm. There was not much dilatation above.

A radium plaque was applied to the neck and under an anæsthetic the œsophagus was dilated to a No. 14 bougie. Great difficulty was experienced in forcing down the smaller numbers through the stricture.

Next day swallowing was easier.

27.2.40 : Dilatation was again performed and swallowing again improved.

There is a possibility that the right vagus may be pressed upon by the enlarged glands and that this is the cause of the spasm.

## Section of Medicine

President—Sir MAURICE CASSIDY, K.C.V.O., M.D.

[January 23, 1940]

### DISCUSSION ON CYSTIC DISEASE OF THE LUNG

**Dr. W. Burton Wood :** Cases of hydatid cysts and closed fluid cysts are very rare conditions in this country. Dermoid cysts are usually of mediastinal origin. The sacs which form the essential feature of the so-called congenital cystic disease of the lung are not cysts according to the meaning of the word in its pathological sense (Murray's and Oxford Dictionaries). I propose, however, to include under the term cystic disease saccular formations which are not closed and which contain air, whether or not fluid be also present. Though fluid-filled cysts of the lung of any kind are very rarely seen, air sacs are relatively common.

It is only in recent years that much attention has been given to cystic disease of the lungs. Thus Sir Thomas Barlow [1] in 1880 recorded his discovery of a cyst in the lung of a child who had died at the age of 3 months. This was a small air cyst, the size of a chestnut, with a smooth lining and a thin wall apparently composed of collapsed air vesicles. No opening to the cyst was found. This was not the first example recorded. A certain Fontanus had described a similar discovery in the seventeenth century. In this instance also the child had died at the age of 3 months, and one lung was found to be partly replaced by an air bladder which, however, was in communication with the bronchi. This was only one of several noted before Barlow's time, but the recognition of cystic disease in the living and the demonstration that the condition is not very uncommon had to await the advent of chest radiology and the introduction of opaque oils for lung delineation.

In the last ten years some 1,500 problem cases have been presented at a clinic for tuberculosis officers held at the London Chest Hospital. Among these cases 20 were diagnosed as "cystic disease of the lung". Again during the period 1934-39, 121 patients were admitted to my beds at Victoria Park suffering from bronchiectatic conditions. Of these 89 were diagnosed as cylindrical bronchiectasis, 19 as saccular bronchiectasis, while 13 were regarded as examples of cystic disease.

The commonest form of cystic disease of the lung is a cystic bronchiectasis, but one which exhibits such striking differences from saccular bronchiectasis that we are compelled to regard it as an entirely separate entity, though sometimes the two forms may be seen in the same lung. Some of the differences emphasized by Sellors [2] may be tabulated as follows :—

	Saccular Bronchiectasis Degenerative	Cystic Bronchiectasis Developmental
Origin		
Distribution in lung	Tends to be basal	Tends to be in upper and middle zones
Associated bronchi	Deformed and dilated	Approximately normal
Relation to sepsis	Accompanies or follows infection	Precedes infection when this occurs
Pathology	Indicates a destruction of normal tissues	Indicates an abnormal growth of normal tissues
X-ray appearance	Not distinctive before injection of lipiodol, irregular saccular blobs at the end of clubbed bronchi in bronchograms	Oval or smoothly rounded fine annular shadows of almost distinctive appearance before lipiodol

## PRELIMINARY SORTING BASED ON X-RAY APPEARANCES

*Balloon cysts.*—A single cyst replaces a large part of the lung field. They may be expansile, sometimes swelling rapidly to fill a hemi-thorax and push over the mediastinum—sometimes diminishing or even disappearing.

*Solitary cysts.*—Smaller in size than the balloons, sometimes expansile, sometimes more than one in a lung field.

*Bubble cysts.*—The grossest type of cystic disease, in which often a whole lung appears to be replaced by an aggregation of medium-sized cavities. (Note the curious resemblance to a picture of air-containing bowel. In the skiagram of an infant's trunk bubble cysts in a left lung and gas loculated in the intestine may be indistinguishable and may suggest a diaphragmatic herniation of the gut.)

*Berry cysts.*—Clusters of smaller cysts disposed in grape-like bunches often confined to a single lobe. These cysts, as outlined by lipiodol, are beautifully rounded. They are sometimes isolated, giving the appearance of a plucked bunch with one or two berries left pendant.

*Fluid-filled cysts.*—An example of a fluid cyst in a negro infant which discharged its contents after rupturing into a bronchus, thereafter becoming converted into a balloon cyst, is given by King and Harris [3], and it has been maintained that all cysts are originally fluid and only become aerated after discharge of the contents.

## CLINICAL GROUPING

*Balloon cysts.*—These form a separate clinical group. They are usually discovered in infants or young children. They are characterized by large size, thin walls, and a tendency to increasing inflation by check-valve action of the supplying bronchus. This may lead to attacks of acute dyspnoea and to signs suggesting a pressure pneumothorax. (In the X-ray picture the absence of a collapsed lung at the hilum and traces of pulmonary markings at the extreme apex or base are valuable evidence when distinguishing from pneumothorax.)

A very good example of a balloon cyst was published by Dr. E. H. Hudson [4] in March 1939. A male infant 6 weeks old began to suffer from attacks of distressing dyspnoea. Puncture through the chest wall to relieve pressure symptoms led to a partial pneumothorax around the cyst-filled lung and confirmed the diagnosis. Unfortunately surgical treatment failed, though only just failed, to effect a cure. An example of balloon cyst in an older child (a girl of 9 years) is described in the *Australian and New Zealand Journal of Surgery* for October 1939. This was successfully treated by total pneumonectomy. An abnormal bronchus was found pursuing a tortuous course in the cyst wall before opening into it and the conditions were evidently suitable for check-valve action.

*Solitary cysts.*—The solitary cyst is usually discovered by accident, nor is there any reason why it should cause symptoms unless infection converts it into a local abscess; or hæmoptysis, a common symptom in cystic disease, sends the patient to a doctor. Such a cyst may, however, cause difficulty in diagnosis. Thus a tuberculous cavity, especially in the subapical region of the lower lobe, if of the thin-walled, exactly spherical elastic type, may closely simulate a cyst. Here, however, serial sputum tests and skiagrams should soon settle the problem.

It may be more difficult to distinguish between an emphysematous bulla and a cyst. The patient's age, evidence of emphysema, and the peripheral situation of the bulla, may assist in the decision, which is usually of no more than academic importance, for an emphysematous bulla is symptomless unless it bursts, and no treatment is necessary.

*Multiple cysts.*—(a) Gross "bubble" type. Cystic disease, even of this order, may give rise to no distinctive symptoms unless or until secondary infection supervenes, though there may be a history of bronchial catarrh or asthma. In some instances there may be a history of pneumonic attacks. Not every "damaged lung" in childhood is the result of interstitial pneumonia—in some cases pneumonia in the first instance may well have attacked a damaged, or rather, a deformed lung. The bearer of a cystic lung is in constant danger of a secondary infection, and when this occurs the results may be devastating, as the pus forms pools in the lacunæ.

A boy of 18 years came to see me because seven weeks earlier he had suddenly begun to cough up pints of pus. His mother said he had been wheezy since infancy, but had otherwise enjoyed fair health and had, since leaving school, worked as a milk roundsman. X-ray examination provided the typical picture of a "bubble lung". He was admitted to hospital where he died within a few days of septic bronchopneumonia and toxæmia. The right lung showed universal cysts and the absence of pigment, which suggested that the lung had never functioned. Microscopic examination of sections showed the appearances typical of cystic disease—to which we shall refer later [5].

I have recently seen two adult patients in whom a fulminating septic infection of an upper lobe was associated with a cystic condition. In one whose right upper lobe was converted into a sac of pus X-ray examination in a lateral position showed pus pockets apparently due to this cause. In the other the cysts were only discovered after death from a lingering septic pneumonia and had not been suspected during life. Dr. Gloyne's sections of the cyst walls confirm the naked-eye impression that these cysts are of developmental type and not ordinary abscess cavities.

Septic infection does not always occur, however, and even a bubble lung is compatible with fair health for indefinite periods.

(b) Cystic disease of the berry type. This often occurs in an upper lobe where conditions for drainage are of course good. The signs of upper-lobe retraction with excavation inevitably suggest a diagnosis of fibroid phthisis, and when hæmoptysis is a prominent symptom mistakes in diagnosis are likely. One such case was referred to me by Dr. Howell (tuberculosis officer for Bethnal Green and Hackney), and he told me that the patient had already been notified as suffering from tuberculosis on about a dozen occasions.

The rupture of a thin-walled superficial cyst may be the cause of spontaneous pneumothorax. One of my patients, a young man whose right middle lobe showed cystic disease of the berry cluster type, developed this complication. It caused him no serious disability but the middle lobe remained in a state of semi-collapse and the local pneumothorax has persisted.

#### PATHOLOGY

The pathology of cystic disease of the lung (of the type we have been discussing) has been admirably described and beautifully illustrated by Holmes Sellors [2] in his recent paper on "Congenital Cystic Disease of the Lungs". My visits to the pathological department at Victoria Park have only confirmed the accuracy of his observations. Sellors points out also that in the cavities of acquired bronchiectasis, although secondary inflammatory changes may be gross, the arrangements of the supporting tissues bear some relation to the normal.

#### ÆTIOLOGY

Cystic conditions of the lung have been found in the foetus, at or soon after birth, and in later life at all ages. They are connected with the bronchial tree, usually communicate with bronchi, and their walls exhibit a bronchial-wall-like structure.

Under the microscope the structure of their walls exhibits evidence of disorderly growth, not of degeneration. The cysts can be differentiated from ordinary bronchiectasis, even from that of saccular type. The cysts appear to result from a failure of the extremely complicated system of lung budding which should proceed until the final alveoli are formed. The budding stops prematurely, whether earlier or later, and the terminations of the already formed bronchi apparently swell out and thin out to occupy the spaces where pulmonary parenchyma should have developed, and dilated bronchi take the place of the lung alveoli.

Why this failure occurs we do not know. But neither do we know why in the immense majority of mankind the extremely complicated process of lung budding proceeds with exquisite precision till development is complete. Other congenital defects, e.g. dextrocardia, have been noted in association with cystic disease, and one of my series showed this deformity.

#### TREATMENT

It is usual to contrast the claims of medical and surgical treatment, but if we are right in assuming a congenital defect, medical treatment must be merely palliative. We shall have in every instance to compare the risk of leaving a cystic lung alone with the risk of attempting its removal. I would suggest that: (1) Balloon cysts in infancy and early childhood should be removed if they are causing symptoms or if they are known to be of expansile type. (2) Cysts of the berry cluster type, especially those of the upper lobe, should not be interfered with. (3) With regard to the cysts of bubble type I believe that removal by lobectomy or pneumonectomy is advisable if the condition is discovered in childhood and before secondary infection has produced a pus-sodden lung. This is not inevitable, but the risks of its super-vention are so considerable and the result of it so horrible, that I feel the hazards of operation are less than those of the disease. When suppuration has occurred operation may be too late, and we are left with such measures as posture and bronchoscopic drainage, or creosote, i.e. with ineffective palliatives.

Exact exploration of the whole bronchial tree by lipiodol must precede any consideration of surgical operation for a disease which more often than not affects both lungs. Removal of a single lung is futile unless its fellow is sound, and a bilateral total pneumonectomy is still beyond the scope, though I can scarcely think it is beyond the ambitions, of a really progressive thoracic surgeon!

I should like to acknowledge the help I have received in preparing this paper, compiled under some difficulty, from Mr. Holmes Sellors, who generously placed at my disposal material collected in his recent study of the subject from which I have freely drawn. I am also indebted to Dr. Lloyd Rusby and Dr. E. H. Hudson for allowing me to use skiagrams of cases under their charge, and also to Dr. D. S. Page, of the Pathological Department of the London Chest Hospital.

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- 4 HUDSON, E. H. (1939), *Brit. M. J.*, (i), 503.
- 5 BURTON WOOD, W. (1934), *Tubercle*, **16**, 49.

**Mr. Holmes Sellors :** The healthy bronchus shows ciliated columnar epithelium, outside which are grouped the supporting tissues, viz. cartilage, muscle, elastic, fibrous, and lymphoid tissues with mucous glands. In the case of bronchiectasis the epithelium shows inflammatory changes with desquamation and even in some

parts ulceration. In the supporting tissues inflammatory infiltration is the dominant note, though their proportion and arrangement are unaffected.

When, however, we come to cystic disease there are two striking features, the one being the regularity and constancy of the lining epithelium, and the other being the erratic distribution and amount of the supporting tissue. Even in large cysts which have been the site of suppuration the epithelium often retains its integrity and orderly character, a point that helps in determining diagnosis in cases which have been treated under a different label. As regards the supporting structures fibrous tissue is the most common feature, but in some instances blood-vessels, muscle and mucous glands predominate to such an extent that tumour formation has been suggested.

The question of mistaken diagnosis is very common in cystic disease. Localized or interlobar empyema is often diagnosed, and the case treated by drainage under that supposition; it is only when the cavity or space shows little or no signs of healing that the possibility of an infected cyst is thought of. In cases of doubt the cavity should be reopened and explored to exclude any mechanical cause, and at the same time a piece of the wall removed for section. If bronchial epithelium is found a cystic origin is probable. Lung abscess is not so likely to cause trouble in differentiation, the surrounding pneumonitis of the true inflammatory condition being marked and the clear-cut rounded cavity of a cyst not being so apparent. If, however, operation for drainage is indicated, it is wise to make a biopsy of the wall.

Pneumothorax is liable to be confused with the large balloon type of cyst, and here the tomograph is of value in showing the continuous outline of the cyst against the chest wall. Association of pneumothorax with a cyst can occur, but it is more usually the aspirating needle introduced into what is thought to be pleural space that produces the double event.

Tuberculous cavities can be difficult to distinguish when there is a negative sputum and absence of surrounding infiltration. It may be mentioned that hæmoptysis is a common feature in the case of cysts. Emphysematous bullæ are similarly confusing, though prolonged observation may show some alteration in the size of bullæ, but not of cysts.

In the case of multiple cysts of lobar or "honeycomb" distribution the differentiation from bronchiectasis is really only of academic importance. If there is purulent sputum or any evidence of toxæmia, surgery is indicated in either case, so long as the condition permits of lobectomy or pneumonectomy. Two cases which were treated by pneumonectomy can be quoted to indicate some of the diagnostic differences. The first showed an irregular distribution of large cavities through the right lung, which incidentally had an azygos lobe. If the condition had been one of saccular bronchiectasis the symptoms might reasonably have been expected to be very much more severe than they actually were.

In the other case the middle lobe and anterior basal bronchi of the lower lobe were affected, while the posterior basal and mediastinal bronchi of the lower lobe were quite normal—a state of affairs that could hardly be envisaged with bronchiectasis as we know it. The histological findings in both these cases showed that they could be classified as cystic disease in contradistinction to infective bronchiectasis.

As regards treatment, the governing factor is the risk of infection if the cysts are left alone. Removal of an uninfected cyst is quite straightforward, whereas excision of an infected or already drained cyst is a much more hazardous task. Many of the cases come under observation in childhood, and a further advantage of surgery may be illustrated by the very definite improvement mentally and physically that follows removal of the diseased area.

[Mr. Sellors showed a series of coloured drawings from microscopic sections

showing the essential points in the pathology of cystic disease when compared with the normal and dilated bronchus.]

**Mr. J. E. H. Roberts** said that as regards one class of specimens which Dr. Burton Wood showed, those of multiple cysts in one lung, with gross deviation of the trachea and mediastinum to that side, these were not cases of cystic disease but of bronchiectasis. It was clear from the examination of lobes and lungs removed by operation that both cystic disease and bronchiectasis could co-exist, as in a specimen which he had there in a jar. The bronchiectasis might be independent, or it might be secondary to the cystic disease, due to pressure upon a bronchus with atelectasis then occurring, followed by bronchiectasis. He had never seen deviation of the mediastinum to the side of the cystic disease in cases uncomplicated by bronchiectasis.

The explanation that these cysts might be due to arrested or abnormal development of the lung buds appealed to him as probably true, and he mentioned two cases of cysts lined with ciliated columnar epithelium which he had operated upon, one in the wall of the lower part of the thoracic oesophagus, and one attached to the posterior part of the trachea, both these probably being due to aberrant lung buds, distinct from the buds from which the normal lungs were developed.

Whatever might be said of operation in adults, in his opinion removal of the offending lung or lobe should be done in children before infection occurred. The operation in children was relatively safe. He had performed nine total pneumonectomies for bronchiectasis and one for cyst in children from the age of 2 to 14 without any mortality.

[Mr. Roberts showed slides of illustrative cases.]

**Mr. Tudor Edwards** agreed that apart from parasitic cysts there were three main types of cystic disease of the lung (1) the multiple, unilateral or bilateral type, (2) the simple unilocular cyst, and (3) the giant cyst.

The uncomplicated cyst, apart from the giant variety, rarely causes symptoms. The giant cyst may cause dyspnoea, particularly on exertion, owing to the displacement of the mediastinum and the pulmonary collapse which they cause when distended.

The most common complication in all varieties is secondary infection; when the cyst is unilocular and has a thick layer of pulmonary tissue between the cavity and the visceral pleura these are often diagnosed as pulmonary abscess, but when the pulmonary layer is thin and airless they are commonly mistaken for empyemas even at operation. They may be diagnosed, before operation or aspiration, by the presence of air in the cyst which can be seen on radiological examination and which demonstrates a fluid level altering with posture. At operation they show the appearance described as "lattice" lung, and it is a reasonable assumption that most, if not all, cases of such a condition, follow the drainage of an infected cyst or a pulmonary abscess and are never found in the uncomplicated empyema.

Another complication is the occurrence of spontaneous pneumothorax, in which no pulmonary abnormality has been previously suspected. In one patient it was possible to induce intrabronchial lipiodol to escape into the pleura through the cyst, when it could be seen radiographically in the costophrenic angle.

There appears to be general agreement about the treatment, namely that the unilocular and particularly the giant cysts should be removed, generally by lobectomy or pneumonectomy, as soon as the diagnosis is made. The previous incidence of infection, especially in the thin-walled giant cyst, will add considerably to the mortality owing to firm adhesions to important mediastinal structures such as the venæ cavae, which if torn, may result in fatal air embolism.

The thick-walled unilocular cysts should, when containing pus, be drained for several weeks before carrying out lobectomy, as this diminishes the risk of "spill-over" to other parts of the lung during the operation with subsequent infected atelectasis. These cases stand operation well and usually recover more rapidly than those undergoing operation for bronchiectasis.

**Dr. J. G. Scadding** said that he thought that the number of cases in which a diagnosis of multiple cystic disease of the lung could be made in adults, with confidence, was very small. In cases in which there was gross infection, so that the symptoms were identical with those of bronchiectasis, the position usually was that the diagnosis must be left in doubt. The only cases in which he felt confident about the clinical diagnosis were the large single cysts and multiple cysts in which there was no evidence of gross infection.

He gave a brief account with illustrative radiograms, of three cases, two of single cysts, and one of multiple "bubble" cysts confined to one bronchopulmonary segment.

**Dr. Philip Ellman** referred to the association of congenital cystic disease of the lungs with cystic disease in other organs. He mentioned a case which had originally been seen by Dr. Clifford Hoyle and which had come out to his Sector of the Emergency Medical Service. The patient was a man aged 56 with congenital cystic disease in the upper zone of the right lung, who had been in sanatoria and treated in the early days as a case of pulmonary tuberculosis.

Radiograms and tomographs showed multiple cysts with horizontal fluid levels in the cystic area. In addition this patient had large lobulated hydronephrotic kidneys with calcification in the cysts. Complete investigations of the kidneys with intravenous pyelography had been done by Mr. Yates Bell.

Another interesting feature in this case was the existence, unknown to the patient, of old fractures of the ribs, and the results of investigation showed that he was a definite case of congenital cystic disease of the lungs with cystic disease of the kidneys and possibly an old inactive obsolete hyperparathyroidism.

Dr. Ellman in a search of the literature had not found any reference to the coincident occurrence of cystic disease in other organs, and asked for the experience of members of the Section in this connexion.

In reply, **Mr. Holmes Sellors** stated there were a few references to cysts found in the fetus, but he thought that a good many more had been seen but not recorded. Several pathologists he had spoken to recalled cases which would have answered to the description of cysts in the fetus, but the specimens had not been retained.

He again emphasized the risk of infection in cysts and said that secondary bronchiectasis of the "spill-over" type was a common late association of the untreated case.

[March 26, 1940]

## DISCUSSION ON THE PROGNOSIS AND TREATMENT OF HYPERPIESIA

**Professor John A. Ryle :** At the time of the Bright centenary celebrations in 1927 [1] I pointed out that the chapters composing the natural history of hyperpiesia, both from the point of view of its clinical and pathological study, were largely written between the years 1827 and 1881 by four distinguished teachers and investigators of the Guy's school—Bright, Wilks, Gull, and Mahomed. Since then modern methods have provided better means of differentiating the clinical picture of hyperpiesia from that of chronic renal disease and of benign or symptomatic hyperpiesis. Advances in respect of prognosis and treatment in the same period have not been conspicuous.

On our prognostic assessments and a broad understanding of natural prognosis the management of cases at various stages of the disease and also the logical appraisalment of measures of treatment must very largely be based. In the therapeutic field the time has come to dispose finally of a number of useless drugs which have been advocated, and to discard certain dietetic and other fads which have too long prevailed.

I have notes of 632 private cases of hyperpiesia, excluding a considerable number filed under the headings of such late complications as cerebral arteriosclerosis with cerebral thrombosis or hæmorrhage and coronary thrombosis or heart failure. The information acquired during the collection of this series, combined with hospital experience and the passage of the years, has provided material enough to allow the formulation of a useful working scheme of prognostic grading.

### *Definitions and Nomenclature*

By hyperpiesia we should understand, as Clifford Allbutt [2] advised, a disease *sui generis*, "a malady", to use his own words, "in which at or towards middle life blood-pressures rise excessively, a malady having a course of its own and deserving the name of a disease". By hyperpiesis we understand rather the fact of high blood-pressure; by hyperpiesia the disease of which high blood-pressure is the most constant and significant clinical feature, a disease having a tendency to terminate, after a period of years, by cerebral hæmorrhage or cardiac defeat, and having for its most constant morbid anatomical findings hypertrophy of the left ventricle and the presence of what Gull and Sutton [3] called arterio-capillary fibrosis and for which Evans [4] more recently preferred the description of diffuse hyperplastic sclerosis.

I would put in a plea for Allbutt's nomenclature in preference to "chronic Bright's disease", as it was originally known, since this involves a confusion with kidney disease which we wish to avoid; to "arteriosclerosis", which is a pathological accompaniment and late feature of the clinical disease; and to "essential hypertension", which is descriptively weak and etymologically unsound, the first word having little more sense than "idiopathic" and the second word being compounded of Greek and Latin roots. Also we are, in fact, more concerned with pressures than tensions. Hypertension, it is true, has gained general acceptance in America, but it is surely preferable that we should be guided in our nomenclature by one of the greatest English authorities on the disease and by one who was ever recognized as a purist in the matter of words and language. The designation "malignant hypertension" applied to a limited group of cases carries unsatisfactory nomenclature a stage further, for everywhere else in medicine—now that malignant pustule and malignant endocarditis have been superseded—"malignant" has come to convey the idea of neoplastic disease.

### Prognostic Grading

In the individual case of hyperpiesia, age, sex, weight, occupation, temperament, mode of life, family history, symptoms, and physical findings—particularly those relating to the number and quality of the secondary consequences of sustained high pressure and arterial change—must all be taken into account in framing a prognosis. The menopausal and the so-called malignant cases require a separate assessment. But the most noteworthy variations in prognosis are naturally those which accompany the several stages of the disease. Of such stages there may be said to be five.

In the *first stage* the patient, a man of perhaps 45–50, presents himself *without symptoms* for a life-assurance examination or a routine overhaul, or for some separate indisposition. He is found to have a blood-pressure, say, of 170/100. Arterial thickening and cardiac hypertrophy cannot be clinically demonstrated and there is no albuminuria. *There is hyperpiesia without arteriosclerosis.* In this stage we may even conceive that the condition is modifiable. The patient may well have ten, fifteen, or more years ahead of him. He may continue his normal activities for five or more years without disability.

In the *second stage* he presents himself for slight symptoms often referable to head or heart and commencing signs of secondary pathology. There may be a B.P. of 200/110; a trace of albumin in the urine; retinal and radial arteries appreciably thickened; a demonstrable left ventricular enlargement and an accentuated aortic second sound. *There is hyperpiesia with arteriosclerosis and cardiac hypertrophy, but there has been no vascular accident or manifest structural damage to any organ.* He may have five years to go before some of these late consequences appear. His total prognosis is not likely to exceed ten years.

In the *third stage* there are symptoms and signs of minor vascular accidents (retinal hæmorrhage, transitory aphasia, or a lesser-grade coronary thrombosis), or of cardiac disability (effort angina, nocturnal dyspnœa). B.P. 220/120. *There is hyperpiesia with arteriosclerosis and early structural damage to viscera.* The total prognosis is not likely to exceed five years and may be shorter.

In the *fourth stage* there are symptoms and signs of gross vascular accidents (cerebral thrombosis or hæmorrhage, coronary thrombosis), or of congestive failure. From such accidents clinical recovery cannot be more than partial and temporary. B.P. 240/130. *There is hyperpiesia, with arteriosclerosis and serious structural damage to viscera.* The prognosis will generally not exceed one or two years.

In the *fifth stage* the patient is bedridden with chronic heart failure, hemiplegia, or the mental deterioration of a more general cerebral arteriosclerosis. There may for the first time be commencing renal failure. The systolic pressure may have dropped but the diastolic figure remains high, with such readings, perhaps, as 190/140. In the presence of heart failure the readings may be much lower. The prognosis is reduced to days, weeks, or at the most, months. *There is hyperpiesia with widespread arteriosclerosis and gross permanent damage to one or several organs.* The prognosis of the so-called "malignant" cases corresponds with that of the fourth or fifth stages of the more chronic or gradual form of the disease.

The prognosis of the menopausal cases is much better than that of male cases at the same age, and progress to the stage of arteriosclerotic degeneration is less likely to occur.

### Treatment

Treatment clearly has its best opportunities in the first and second stages, and after the third stage is almost powerless to affect the duration of life although it may modify disabilities.

Drugs may be useful for secondary symptoms, as in the case of nitrites for anginal pain, morphine for cardiac pain and nocturnal dyspnœa, and digitalis for arrhythmia, but for the hyperpiesia itself they are of little avail. Evans and Loughnan [5] have made a valuable contribution to practical medicine in exposing, by clinical experiment, the futility of a large number of drugs which have from time to time been advocated

and extensively used. I have never, personally, used any of them except the sedatives (bromides and barbiturates), and these have generally been reserved for cases in which nervousness or anxiety seemed to be playing an effective part by preventing restfulness of body and mind. Such nervousness and anxiety can also be helped by sensible explanation and reassurance. They seem not only to contribute to, but sometimes to be aggravated by, the physical changes incidental to the disease, for changes of temper and emotional behaviour are very liable to develop in the cerebral arteriosclerotic. At no stage should we allow our patient to acquire too keen an interest in his blood-pressure readings or report them regularly to him. We stress the contribution to his own well-being of an elimination, whenever possible, of anxious or worrying work and affairs without permitting him, before it is necessary, to lapse into the invalid life. We make capital of his phases of improvement in response to a better ordering of his days and, for as long as possible, protect him from knowledge of such anxieties as we may ourselves be forced to entertain. As a profession we have erred far too often in the direction of pessimism in hyperpiesia and have attached undue importance to the mercury level as compared with other clinical evidence.

Of all the physical aids in hyperpiesia reduction of weight in the obese patient (as was emphasized by Allbutt [2]), is probably the most important. A drop in weight and a considerable fall in pressure are sometimes accompanied by a striking symptomatic, albeit temporary, improvement. I know no physiological justification for the strict curtailment of red meats, milk, and salt, which are often enjoined. In heavy patients I have thought a reduction in fats and starches more reasonable and more effective, presumably by a simple lightening of the load to be carried by the heart and muscles, and also, perhaps, of the metabolic load. On general principles, since both are tissue-poisons, rather than because they are suspected of specific action, alcohol and tobacco should be very moderately used. It has yet to be shown whether the sex-hormones have a place in the treatment of menopausal hyperpiesia.

In the first and second stages there is no objection to moderate open-air exercise, which is beneficial to the sense of mental and physical well-being alike. Both in the earlier and later stages instructions in regard to physical rest appropriate to the needs of the individual case are necessary. An hour a day, a day a week, and one week in each three months in bed is a simple injunction which I have often given to patients in the second and third stages and earlier part or better phases of the fourth stage of the disease.

In brief we shall have to agree that we have no specific treatment for hyperpiesia. At present we are insufficiently instructed physiologically in regard to the part played by pressor substances to be in a position to provide an antidote. If an antidote is discovered its utility will presumably be confined to the first and second stages. Surgery, whether of the sympathetic or the renal capsule, is still in the experimental stage. As in other chronic maladies sensible management, simple explanation, and reassurance remain to us. In most chronic diseases management is to be preferred to remedies. Uncensored commercial enterprise and the too-ready advocacy by medical men of preparations lacking all therapeutic trial have done and continue to do a great disservice to medicine.

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**Professor J. Paterson Ross:** *The results of surgical treatment of arterial hypertension.*—The value of surgery in the treatment of hypertension is difficult to assess because there is no standard operation for this condition, and because surgery has been practised for only a comparatively brief period. Even when hypertension

is attributed to a given cause there are variations in technique to be taken into account ; but the chief reason for the diversity of operations is the number and variety of the theories which have been put forward to explain the fundamental cause of essential hypertension, the operations having developed as the notions about their true object have altered.

(1) *The theory that the vasomotor centre is overactive.*—This theory led naturally to operations for resection of the splanchnic nerves. As soon as it was realized that vasoconstriction is not confined to the splanchnic area the operations were extended to include excision of the sympathetic trunks and even section of large numbers of spinal nerves. Since it has been shown that the vasoconstriction is not of neurogenic origin, these extensive and often mutilating operations have become obsolete, though splanchnic neurectomy survives, as it is now performed with a different object in view.

(2) *The theory that the adrenal glands are overactive.*—This theory is translated into fact in the cases of paroxysmal hypertension associated with certain tumours of the adrenal medulla (phaeochromocytoma). Apart from these rare cases, in which removal of the tumour effects a cure, there seems to be nothing to support the idea that excess of adrenalin is a factor in essential hypertension, and partial adrenalectomy cannot therefore be advocated on that ground.

There is more, however, to be said for removal of the adrenal cortex, since hypertension cannot be produced experimentally if the adrenal cortical tissue has been completely excised beforehand ; and experimentally induced hypertension can be abolished by removing both the adrenals. It may be added, however, that in animals it is possible to produce sustained hypertension even after removing one adrenal completely and destroying the medulla of the second—an indication of the importance of the cortical rather than the medullary portion in this connexion.

The danger of total adrenalectomy in man forbids the employment of this method of treatment at present.

(3) *The theory of renal ischaemia.*—In considering this theory from the surgical standpoint it is essential to separate the cases of renal involvement into two distinct groups :—

(a) *Those in which there is some structural abnormality or disease of the kidney.*

(b) *Those in which the disease is confined to the renal vessels, the kidney tissue being apparently unaffected, or being affected subsequently.*

(a) Striking results, usually complete and apparently permanent cures, have followed nephrectomy in the first group. The records include developmental abnormalities such as "atrophy" and ectopia of the kidney, infarction, renal damage secondary to calculus, or obstruction of the ureter, and also pyelonephritis. In all cases of hypertension, therefore, it is essential that a thorough urological examination should be carried out to search for evidence of unilateral renal disease, since nephrectomy may cure the condition rapidly and completely.

If for any reason nephrectomy cannot be undertaken, decapsulation of the kidney should be considered, for it now seems probable that the cause of the increase in blood-pressure in both groups is really the same, the organic kidney lesion in this group compressing or obstructing the renal vessels.

(b) Turning to the second group, in which the primary fault lies in the vessels themselves, the original cause of the vascular change (hyalinization of the glomerular afferent arterioles) has yet to be discovered, and it has still to be proved that this change can produce sufficient renal ischaemia to give rise to hypertension, just as Goldblatt's clamp does in the experimental animal. It has been suggested that spasm of the renal arterioles may be a factor in the development of this primary renal ischaemia, and the effect of sympathectomy has been likened to that of removing Goldblatt's clamp. It was to this idea I referred when I said that splanchnic neurectomy is now undertaken with another object besides the production of vasodilatation in the "splanchnic area".

I have had the opportunity of performing splanchnic neurectomy on only ten patients, the first three and a half years ago; it is therefore too early to say whether life may be prolonged by operation. In one patient I divided the nerves above the diaphragm, in all the others below, since this approach enables one not only to reach the nerves quite simply, but also to inspect the kidney and adrenal and to remove a portion of the lumbar ganglionated trunk if this is considered necessary.

Of the 10 patients seven derived considerable benefit from the operation, their symptoms (particularly the headache) being relieved and their activity restored almost to normal. The symptomatic relief has persisted in spite of a return of the blood-pressure almost to the pre-operative level in three patients; in three the pressure has remained near the post-operative level, and the last one was operated upon too recently (four months) to tell what his pressure may do.

Two patients derived little or no benefit, and I regret to say that one died a few days after operation perhaps because, contrary to the usual practice, I decided to operate on both sides at the same sitting—the sudden fall of pressure may have been too much for him to stand, though an unexplained infection was also a factor.

I have found it extremely difficult to judge beforehand either from examination of renal function, the ophthalmoscopic findings, or the results obtained by administering vasodilator drugs, what the operation is likely to achieve. I must add, however, that I obtained the most striking success in this small group in a patient upon whom I felt inclined to refuse to operate—she was 44 years of age, with a history of only fifteen months, a pressure of 270/190, very severely damaged kidneys and well-marked visual impairment with hæmorrhages and exudates in both fundi. I feel, therefore, that so long as we are satisfied that in spite of the best constitutional and medical treatment the disease is progressive and that the patient is incapacitated and in pain, the chance of surgical relief should not be withheld, at any rate until we have more reliable pre-operative tests.

Since it is sometimes stated that "any operation" will lower the blood-pressure, and that sympathectomy has no special value, I must say, in passing, that several of my patients had had previous operations—one had cholecystectomy and two had hysterectomy performed since the onset of symptoms of hypertension without any appreciable effect on the blood-pressure.

If we may assume that vaso-spasm can give rise to renal ischæmia, and furthermore (in view of our observations of prolonged vaso-spasm elsewhere) that spasm may in time produce structural disease of the affected vessel walls, it is clear that if sympathectomy is to succeed it must be done as early as possible. So long as Goldblatt's clamp remains on the renal artery sympathectomy is of no avail; only spasm could be relieved by sympathectomy; structural disease of the vessels resembles the effect of a permanent clamp. The prognosis of operation must therefore depend (as in all other vascular diseases treated by sympathectomy) upon the severity of the disease in the particular case, and especially upon the presence of structural changes in the vessels.

Splanchnic neurectomy may yet, and perhaps soon, be superseded as the accepted surgical treatment for hypertension. If the abolition of spasm in the renal arteries be the real object of the operation, should we not perform renal sympathectomy instead of the more elaborate splanchnic neurectomy? It would certainly entail a post-ganglionic section, which might not prove ultimately to be as good as the pre-ganglionic operation, but that remains to be shown by experience. Further, it is important to recall the experiments of Cerqua and Samaan who cured experimental renal hypertension by subsequent decapsulation of the kidneys. Already there is some clinical experience to support their experimental evidence, but much more will be required before a verdict can be given upon the value of surgery in the treatment of hypertension, though operation may relieve symptoms which have not yielded to simpler forms of therapy.

## Section of Laryngology

President—T. B. LAYTON, D.S.O., M.S.

[March 1, 1940]

### DISCUSSION ON THE INDICATIONS FOR REMOVAL OF TONSILS AND ADENOIDS IN CHILDREN

**Sir Lancelot Barrington-Ward :** The term "tonsils and adenoids" is used rather loosely for a common condition in children without differentiation between the relative importance of tonsils and adenoids. In my experience, the tonsil is the more dangerous element. Its structure allows organisms to lie dormant, to remain moderately but continuously active leading to chronic general poisoning, or to flare-up into acute inflammation at indefinite intervals. Adenoids, on the other hand, only act mechanically by obstructing normal respiration or by a low-grade infection causing inflammation of the ear or certain cervical glands. The importance of adenoids is greatest in the earlier years of life and they may require removal within the first few months. In later years, if adenoids are present, the tonsils are usually infected as well, and it is wise to deal with both. If adenoids only are removed and septic tonsils are left behind adenoids are very likely to grow again.

What are the pathological conditions of the tonsils which demand operative interference? The most usual description on a case sheet is that they are large and septic, but this combination is not always constant. Large tonsils may not be septic and septic tonsils may not be large. It is true that sepsis often determines hyperplasia of lymphoid tissues, but it is not the only cause.

Mere size of a tonsil is not an indication for its removal, unless by reason of its size it is injuring the patient. Sometimes a persistent cough, with all other causes excluded, may justify interference, but this is rare. More commonly there is actual obstruction to the food and air passages. A septic tonsil, whether large or small, is a menace to its owner, and should always be removed. How are you to decide that a tonsil is septic? It is impossible to describe a standard appearance in health and disease that will satisfy every observer, and bacteriology is not always helpful. More is learnt by the study of the secondary effects. Some of these are more direct than others.

#### *Direct*

(1) *Repeated tonsillitis.*—If a child suffers from sore throats from time to time, with or without fever, it is wiser to take a long view and realize that the only certain way of stopping further attacks is to remove the tonsils. The tonsil is low-grade tissue with a poor blood supply, with little power of recovery, and difficult to disinfect. Organisms lie latent in its recesses for long periods of time, as is well shown in the diphtheria carriers. I know of many an adult who has regretted that the indications for tonsillectomy in his childhood had been neglected. Tonsillitis in children has

certain peculiarities. It is not always easy to diagnose it by symptoms alone. Children often do not complain at all of the throat, even during a sharp attack, and, unless a routine examination of the throat is made in every sick child, it can easily be missed. It is one of the commonest causes of unexplained pyrexia and it is a frequent cause of abdominal pain.

(2) *Chronic enlargement of the upper deep cervical lymphatic gland* (Wood's tonsillar gland) on either or both sides of the neck, without enlargement of other glands, always indicates a tonsil infection. Such a gland is very likely to receive and harbour the tubercle bacillus of bovine strain, and in some instances the tubercle bacillus can be found in the tonsil itself. In the treatment of tuberculous glands, whether radical or conservative, removal of the tonsils is an essential stage.

In the experience of all of us, the incidence of tuberculous glands has shown a remarkable reduction in the last twenty years. I am sure that the improvement is due more to the careful examination and treatment of throat conditions in school children than to the improvement in the purity of milk.

(3) *Otitis media* with its sequences, mastoiditis and deafness, are in the majority of instances the result of nasopharyngeal catarrh secondary to infected tonsils and adenoids. Here again we see a great advance in recent years, due to the continuous medical supervision of the school child and early treatment of this source of infection.

#### *Indirect*

In some the tonsil can be blamed only as the portal of entry of a specific organism and to remove the tonsil after the damage has been done is useless. It is only reasonable to perform tonsillectomy in such a case if there is continued infection or reinfection. Rheumatism with its associated conditions, heart disease and chorea, is a good example. The responsibility of selection lies here with the physician, but the surgeon is permitted to take an intelligent interest in the decision. There is some evidence that rheumatism is less likely to attack individuals who have undergone tonsillectomy, but the influence of the tonsils in precipitating a relapse is even more striking and more easily proved. Schlesinger, in his Milroy Lectures of 1938, in discussing rheumatic relapses, stated that of 67 cases relapsing, 37 were due to tonsillitis and 13 to pharyngitis. It is only fair to mention that two other relapses were due to tonsil and adenoids operations, but this does not reduce the importance of the tonsil because operation on the tonsils must intensify the immediate risk of septic absorption. Nephritis is another disease which in many cases is secondary to tonsillitis, and although grave damage has already been done further injury can be arrested by timely attention to the source of infection.

*Chronic sepsis* may have a wider effect. It is not uncommon to see a flabby child with scoliosis, knock-knee, or valgus of the feet, suffering from chronic infection of the tonsils, and the first part of the orthopaedic treatment must be elimination of the toxæmia. Indeed, any child of poor nutrition and stamina may suffer from such a condition as enuresis, which is cured by restoring the general body tone, and it may be that infected tonsils are playing their part and require attention. Again, in asthma, although an allergic basis is responsible in most instances, allergy will not explain all, and a factor in some is undoubtedly a septic focus such as the tonsils.

The least definite of all indications, and perhaps the one that has brought most disappointment with the operation, is recurrent or persistent nasopharyngeal catarrh and the common cold. If a patient is subject to colds, and if every cold starts with a sore throat, there is reason to remove the tonsils in the hope of preventing further attacks. Experience shows that this is successful in the majority of cases, but complete immunity cannot be guaranteed. In the more frequent form, where the child every winter develops a persistent stuffy catarrh of the nose and throat, it is

possible that the sinuses are affected, or there may be some general metabolic fault and tonsillectomy is useless.

**J. D. Kershaw :** In 1933, when I was Assistant M.O.H. in Ealing, I carried out an investigation among the school children of that borough with a view to establishing a definite code of indications for tonsillectomy. Surveying the children who had been tonsillectomized during the preceding few years I found that persistent and recurrent sore throats, excessive susceptibility to colds, cervical adenitis, and certain other conditions were constantly relieved by the operation, but the investigation of approximately a thousand children who had had notably enlarged tonsils and had not been operated upon showed that tonsillar enlargement *per se*, even when associated with these symptoms in a moderate degree, showed a tendency to spontaneous cure with increasing age in some 90% of all cases.

As a result I laid down a set of guiding principles which are, I believe, still used in Ealing. They are based on both the presence of symptoms and the size and appearance of the tonsils, but, like all guiding principles in medicine and surgery, they must not be too rigidly applied. Briefly, they run as follows:—

Conservative treatment should be tried on the small tonsil with mild symptoms of associated conditions, on the moderately enlarged tonsil with slight symptoms, and on the large tonsil with no symptoms. Operation is indicated upon a small tonsil associated with severe symptoms, on a moderate tonsil with mild symptoms, and on a large tonsil with slight symptoms. In case of doubt, conservative treatment should be tried, but the possibility of later operation kept in mind, while, where the environment is such that conservative treatment is not likely to be persevered with, there must always be a bias in favour of operation.

In 1935 I went to Accrington as the first full-time M.O.H. of the borough. I had expected the dirt and humidity of the Lancashire atmosphere to favour nasopharyngeal disease, but the tonsillectomy rate which had been obtaining in the town was staggering. Some 5% of the school population underwent the operation annually, and assuming the average school life of a child to be ten years, no child had more than an even chance of retaining its tonsils up to the age of 14.

I therefore began a more intensive study of the younger school children. Accrington possesses a nursery school, so that children may begin their school life at any age between 2 and 5, and I have constantly found that between three and six months after the start of school life, at whatever age this may begin, the child's tonsils enlarge and he becomes subject to frequent colds, with or without chronic rhinorrhœa. In from one to two years, depending upon the general physical condition of the child and independent of his age, there is a subsidence of these conditions in about 90% of all cases.

The first intensive exposure of the child to respiratory infections causes a constant series of such infections, to which the natural response of the tonsils is to enlarge. The development of a degree of immunity causes a subsidence of the condition and it is probably not unreasonable to assume that the tonsil plays some part in the development of the immunity, since the few cases in which tonsillectomy has been performed during that stage have shown a tendency to lose their symptoms rather more slowly than those who have not had the operation. In any case, until our knowledge is materially increased, I would suggest that operation during this stage of physiological response to environment is contra-indicated.

Tonsillar enlargement may, like other forms of lymphoid hypertrophy, be in many cases a secondary condition. For the past two years it has been my practice, when a child with enlarged tonsils is submitted to me for examination, to look for signs of chronic rhinitis, turbinate engorgement, or septal deflection, and to attempt some conservative treatment of these. I find the silver proteinate pack to produce marked

alleviation of the symptoms and, in some 60% of my cases (which, admittedly, form a short and unfinished series), a reduction in the size of the tonsils. I am convinced that this question of secondary tonsillar enlargement is worth further investigation, and would like some of my colleagues in the School Medical Service to turn their attention to the matter.

Legally, no child may be operated upon through the School Medical Service without the approval of the school medical officer. The private practitioner cannot, as he sometimes does, demand that operation should be carried out, at the local authority's expense, upon any child whom he considers to need it. At the same time the private practitioner has a perfect right to refer, or to advise parents to take children to the school clinic, and his co-operation is to be sought and encouraged. The school medical officer must, therefore, insist that while he is prepared to accept cases on the recommendation of the private practitioner, such cases must be left to his absolute discretion as regards treatment. Probably the best method for all children whom any assistant medical officer considers suitable for tonsillectomy is, that they should be referred to a senior assistant with special experience, or to the school medical officer himself if he is sufficiently in touch with clinical work.

The consultant to the Education Authority is generally a man who works for the authority during occasional sessions only. His function should be that of a consultant and operating surgeon, in that cases referred to him should be only those where the responsible medical officer feels genuine doubt or considers that operation is justifiable.

The School Medical Service has unlimited material for investigation by the staff, with every opportunity to follow up cases.

**T. S. Rodgers** said that he believed there was only one constant indication for tonsillectomy, namely, repeated attacks of tonsillitis from which the patient did not recover completely in the intervals. Repeated attacks of tonsillitis not due to epidemics were the most important factors in the history, and tonsillitis in children was often a painless disease. The recovery of pathogenic bacteria from the tonsil was of no importance. A recent investigation at the School of Hygiene on the bacterial flora of the nasopharynx had shown that in from a tenth to a quarter of healthy people such pathogenic bacteria were present.

When adenoids were found there was one indication for removal, and that was nasal obstruction which would not yield to conservative treatment. Adenoids he believed to be a result and never a cause of infection. He would take out the adenoids only when there was present nasal obstruction for which no other cause could be found. The characteristic symptoms of adenoids were local. No one should have his tonsils removed before school age, and the older the better. No operation should be done in the epidemic period of the year, the winter.

In every case certain preliminary investigations and treatment should be carried out. The hygiene and bacterial environment of the home should be inquired into, for the child might be living in an overcrowded home exposed to repeated infections. Convalescence in such cases was essential. The teeth, nose, and sinuses, if unhealthy, should be treated before operation, and nose-blowing and nose-breathing established. In one investigation, out of 111 mouth-breathers the mouth-breathing was found to be due to adenoids in only 17. Provision for post-operative treatment was necessary.

There were certain common conditions for which tonsillectomy was done needlessly, sometimes even with harmful results. These were colds in the head, diseases of the nose and sinuses, pulmonary disease, rheumatism, and nephritis. In all these conditions investigations had shown that the tonsillectomized children were in no

better case than those who had not had their tonsils removed; quite frequently the tonsillectomized suffered more.

Tonsils were said sometimes to be removed for the prophylaxis of infectious disease. This he believed to be a fundamental fallacy since immunity to infectious disease was a specific immunity and did not depend upon the state of health. The idea of focal sepsis was now much less widely held than it used to be, and was ill-supported by the facts. Focal sepsis and the diseases to which it was said to give rise were very poorly related. Large numbers of people had obvious foci of sepsis but were free from these diseases, and many who suffered had no such septic foci. Also, the routine removal of septic foci had given poor results, though everybody had met the exceptional case in which sciatica, for example, was dramatically cured by the removal of a septic focus; but then there had been dramatic recovery from sciatica after so many other forms of treatment.

It was estimated that 200,000 tonsillectomies were done each year in England, and there were 85 deaths from it in 1937. There were no accurate figures on the incidence of complications. Keen (1933) had suggested that one in every 20 cases had an interrupted convalescence, and when the speaker was a house physician at Great Ormond Street one of the commonest conditions in his ward was bronchitis and pneumonia following tonsillectomy.

The mere passage of time would cure many patients on whom tonsillectomy might otherwise be done. In London 11,000 children were re-inspected in 1938 some six months or more after being referred for treatment of tonsils and adenoids. About a quarter of these children were found to have recovered without operation.

Conservative treatment and an open-air life had produced remarkable results. In Manchester in 1938 only 60% of the number of operations which had been done the year before were carried out, yet in that year there were 400 fewer cases of ear disease and the children's health continued to improve.

It was a common experience in the L.C.C. children's hospitals for a large proportion of the children admitted with enlarged tonsils to cure themselves without special treatment in a month or two. The other day he examined 300 children in a camp on the East coast and found no single case of enlarged or unhealthy tonsils.

A very strange thing was the wide disparity in operation rates in different localities, a disparity which could only be due to differences in medical opinion; in no other way could it be explained. In one London district the proportion of tonsillectomies rose from 2% of the child population to 25%, the rise coinciding with the appointment of a new medical officer.

The function of the tonsil he believed to be unknown, though it might serve to defend the body against bacterial attack.

In 1913 Professor Burger of Amsterdam said that in his opinion the only indication for tonsillectomy in children was repeated tonsillitis and that judgment, he thought, remained good.

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**Josephine Collier:** The criticism rightly accorded to indiscriminate tonsillectomy is based largely on comparison of the general health and freedom from infection of groups of children who have and have not had their tonsils removed. I do not want now to spend time on some fallacies in these arguments based on mass statistics but I must mention an incidence of 50% of tonsillitis in one series among those who had had a tonsil operation, which does suggest that the operation

has not always been satisfactorily performed. This is not always accounted for in the statistics. But for our present purpose we must grant that tonsillectomy has in the past been performed for inadequate reasons. Is this the explanation of the discrepancy in opinion on the value of the operation? The general practitioner and the children's physician who send for tonsillectomy children whose ill-health after careful consideration and exclusion of other conditions they find associated with diseased tonsils and adenoids are more likely to see good results from the operation than the medical statistician who examines the records of large numbers of children, many of whom have had their tonsils removed because a doctor on routine examination of healthy children has reported the tonsils as enlarged.

The responsibility of the laryngologist in this matter is great. In general the final decision is his, and in his teaching capacity he influences the rising generation of practitioners. For these reasons and because changes in therapeutic fashions are slow in developing, it is important that the indications for this operation should be simple and precise.

Our views on the physiology of the tonsils do not give much practical help in making a decision. We know that this ring of subepithelial lymphatic tissue differs from ordinary lymph glands in having no afferent lymph vessels. Otherwise it behaves like lymphatic tissue elsewhere. The germ centres, now more accurately called "reaction centres", do not develop in animals bred in a sterile environment but appear only when bacteria and toxins invade the organism. For this reason we conclude that protective antibodies are produced in the tonsils. But this defence function occurs equally in all the lymphatic tissues which are particularly active during the early years of life, as is well seen in the compensatory hypertrophy of the lingual tonsil and the lateral bands so often found in patients whose tonsils have been removed at an early age.

At this stage I must join issue with the President in his insistence that the full complement of lymphatic tissue is more particularly necessary for the children of the very poor whose hygienic surroundings are bad. For eight years I was in charge of an ear and throat department of a children's hospital whose patients came almost exclusively from the slums of North Kensington and Notting Dale. Patients for tonsillectomy were sent by medical officers of neighbouring welfare centres where they were under the care of the same doctors for lengthy periods before and after operation. Operation was performed only when the medical officer and I were both satisfied that the child's symptoms were due to the tonsils or adenoids. We had the patients in hospital one or two days before operation, and for a week afterwards, and we never operated during the winter months, during epidemics, or if the child's temperature was above 99° F. With these safeguards we never found any case of deterioration in health.

The chief problem for the laryngologist is to establish a relationship between the state of the tonsils and adenoids and the symptoms or disease from which the patient is suffering. It is useless removing adenoids for nasal obstruction if this is due to enlargement of the turbinates or nasal discharge, either of which may be secondary to infection of the nasal sinuses. Many children breathe or appear to breathe through their mouths without any organic nasal obstruction, and need breathing exercises rather than a nasal operation, which alone will not restore nasal breathing.

I would like here to make a plea for the abolition of the term "enlargement of tonsils and adenoids". The amount of adenoid tissue is of no importance without reference to the size of the nasopharynx; if adenoids block the airway or the Eustachian orifice then they should be removed. With tonsils, too, size is an indication for operation only when obstructive symptoms are present. Occasionally in young children prominent tonsils projecting into the oropharynx or in contact

with the posterior pharyngeal wall, interfere with swallowing sufficiently to give the child a disinclination for eating that hinders his normal nutrition. Removal of tonsils in these patients is quickly followed by normal feeding and improved nutrition. Apart from this type of case these enlargements should be regarded as protective and normal until a condition has arisen which suggests that the tonsillar tissue has itself become attacked by micro-organisms and is so involved that attacks of acute infection are recurring or the tonsil is acting as a source from which distant organs are being infected. An instructive commentary on this aspect of the tonsil problem has been furnished by the medical history of the Basque refugee children who came to this country in 1937. On their arrival the majority of the children, aged between 5 and 15 years, were noted by Dr. Ellis and Dr. Audrey Russell [2] as having tonsils as large or larger than walnuts. Less than 2% had had tonsillectomy but the incidence of both otorrhoea and cervical adenitis was only 0.4% and respiratory infections and nasal discharge were very low. Since their dispersal in different parts of the country their state of health has compared very satisfactorily with English children under the same conditions. A few have developed catarrhal conditions of the nose and tonsillitis is occasionally reported. In examining the records of those who have had tonsillectomy in this country I find there is a certain geographical distribution which points to a variation in medical opinion rather than to any exact relation between symptoms and operation.

Routine removal of tonsils has been advocated for rheumatic conditions, nephritis, asthma and bronchitis, and general malnutrition, and like most routine medicine has produced disappointing results. Statistics give contradictory testimony. In rheumatic children the tonsils should be removed only when tonsillar infections are a feature of the disease. In these cases the intimate association of throat infections and exacerbations of rheumatism is sufficient warrant for tonsillectomy, particularly when the cervical glands remain enlarged between the attacks of throat infection. "Enucleation of tonsils certainly tends to stop severe recurrent throat infections, which on each occasion lay the rheumatic patient open to a recrudescence of the disease" (Poynton and Schlesinger [3]).

Nephritis is an even more difficult problem. Even when history indicates a relation between tonsillar infection and the onset of nephritis, removal of the tonsils does not appear to influence the course of the disease. It has been suggested that the original infection may initiate changes in the kidney which proceed unaffected by removal of the focus from which it started (Addis [1]).

Bronchitis and pneumonia appear from statistics to occur more frequently in children whose tonsils have been removed. My own practice with patients with chronic bronchitis is to remove tonsils only when there is a definite history of tonsil infection. If this is absent, I do not believe prophylactic removal of the tonsils will benefit the patient.

There are signs now that the pendulum is swinging from routine tonsillectomy to routine treatment of supposed nasal sinus disease, and children with frequent colds and recurrent bronchitis are subjected to nasal medication with, or without, antrum drainage, sometimes with less justification than the former wholesale removal of tonsils and adenoids. Undoubtedly the absence of sinus infection should be established before any operation on the tonsils and adenoids is undertaken. In the presence of sinus diseases lavage alone may not cure the condition if adenoids are interfering with the normal ventilation of the nose. But the main problem for the laryngologist is not the child with disease of the nasal sinuses, nor the child with obvious tonsil infection. Treatment of those conditions is simple. The real difficulty is the child whose nasal sinuses are healthy, whose tonsils and adenoids have given no indication of culpability, who yet persists in catching cold after cold. Every

winter he has one or two attacks of bronchitis. After we have decided that some are allergic, we are still left with a number for whom the doctor or the parents demand tonsillectomy. Is this the right treatment? For myself I believe that these are the cases that have brought a useful and necessary operation into disrepute, and I operate only on those with some positive evidence of abnormality.

Removal of tonsils and adenoids is not a panacea for all the ills of childhood, but with proper discrimination in the selection of cases few operations give better results. Each patient should have a well-considered judgment on the relation between the condition of his tonsils and adenoids and the symptoms from which he is suffering. A complete history from the doctor who has been in charge of the patient should be obtained wherever possible, and I welcome the co-operation advocated by Dr. Kershaw.

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**Musgrave Woodman** said that sufficient attention had not been given in the opening papers to the local condition of the tonsils in relation to operation. Mere size had nothing to do with the question. He attached great importance to deep sepsis, and his own method was to use a curved probe pressed on to the tonsil to ascertain its fixity or otherwise.

One of the openers had said that children should never be operated on until school age. Is it not true that the time of the operation should be governed entirely by the incidence of symptoms? The speaker quoted a case of a child with a quinsy at 9 months old which nearly proved fatal.

Dr. Rodgers had suggested that operations on the tonsils for the relief of chronic nephritis were useless. This is not the experience of departments working at the large general hospitals, where many cases of nephritis were submitted for operation with satisfactory results.

It had been stated that a lung abscess frequently followed tonsillectomy. Although this appears to be true in America, it is not the case in this country. A statistical investigation by Dr. Featherstone, honorary anaesthetist to the Birmingham General Hospital, covered all cases submitted to anaesthesia in the hospital during a period of four years, and not a single case had developed a lung abscess or pneumonia following tonsillectomy.

Another statistical fallacy consisted in massing together all operations on the tonsils without reference to the completeness or incompleteness of the removal, and without allusion to the simultaneous presence of sinus disease.

The infrequency of complications in Basque children, living on the sunny slopes of the Pyrenees, who had never had tonsillectomy performed, cannot be compared with children living in a suburb of London, who had had the operation. A very critical discrimination had to be used before statistics were taken as proving anything. Often the argument drawn from the same set of statistics might, with equal facility, be turned in the direction of the inclination of the surgeon.

**C. A. S. Ridout** said that he thoroughly agreed with Mr. Musgrave Woodman concerning the need for a critical attitude towards statistics. In the course of twenty years at his hospital thousands of tonsillectomies had been done, but they had never had a case of lung abscess following the operation, and owing to their follow-up system they would have been certain to have known of it if it had occurred.

It was important also to bear in mind the nature and extent of the operation which was done. In some cases when the child was brought up for tonsillectomy the mother said, "But, doctor, he has had his tonsils removed before".

He agreed that these cases in which there was sinus trouble should always be carefully examined and the trouble removed. Very often it could not be stated whether there was sinus trouble until the patient was actually on the table.

He shared Sir Lancelot Barrington-Ward's view that each case must be considered on its merits.

**M. Hajek** said he could not find any proof of the alleged connexion between tonsillitis and sinus infection. That tonsillar infection was the cause of pathological conditions could only be proved by keeping the tonsillectomized child under observation for some years.

Dr. Hajek mentioned a series of cases where acute or chronic attacks of rheumatism appeared whenever the patients developed tonsillitis. After tonsillectomy there were no such attacks, even the arthritic changes in the joints disappearing to some extent. An example of a different series was that of a woman aged 30. She had an annual attack of subacute nephritis preceded by acute tonsillitis, the nephritis lasting for about six weeks. Although there were no definite signs in the tonsils they were removed. Two days later there was a fresh attack of subacute nephritis lasting for six weeks. But for the next ten years, whilst under observation, there was no recurrence.

He went on to say that in Vienna they saw very few pulmonary complications after this operation. It was done under local anaesthesia in adults and under light ether narcosis in children. A deeper narcosis might cause aspiration of blood and lead to lung abscess.

**W. H. Bradley** said that he believed the tonsillectomy rate in public schools quoted referred to tonsillectomies done before the child arrived at the school and not afterwards. The Medical Research Council inquiry in this matter certainly referred in the main to tonsillectomies carried out before arrival at school. The impressions of those who worked in operating theatres were likely to be different from those of school medical officers or clinicians who looked after school children. He thought that the President of the Section (Mr. Layton) who, unlike the prophet, was not without honour in his own country, was one who could in his position and with his authority assemble the facts and find out the truth of the whole matter.

**R. Scott Stevenson** said that a speaker had suggested that it was the removal of the tonsil itself which caused pulmonary conditions such as chronic bronchitis. He thought that this might rather be ascribed to the inhalation of infected blood-clot at the operation. Unlike his colleagues, he had had one case in which lung abscess did follow tonsillectomy, and that was in an institution devoted to children partly with rheumatism and partly with pulmonary disease. This child had a slight rise of temperature and a cough about a week after operation, without any more serious symptoms, but as it was an ordinary daily routine to screen several chests, the child's chest was X-rayed and lung abscess found to be present. It cleared up with the use of the bronchoscope and postural drainage. He thought that a few cases, if routine screening took place a week after operation, would be found to have something wrong in the chest, but he did not think it was due to the actual loss of the tonsil.

**Sir Lancelot Barrington-Ward**, in replying on the discussion, said that apparently two conclusions emerged. The first was that the school medical officer must in future be a highly trained clinician, and the second was that everybody seemed to accept the view that repeated tonsillitis was the most definite of all indications.

**J. D. Kershaw** (also in reply) said that of the large number of cases of chronic otorrhoea which attended school clinics some 90% responded well to insufflation and ionization treatment. Many of the remaining 10% had already had their tonsils removed.

While agreeing that cases of emergency might arise at any age, he felt strongly that tonsils should not be removed until after the stage of "immunological enlargement" and that the operation should not, therefore, be carried out during the first two or three years of school life.

#### CINEBRONCHOGRAPHY

**Flight-Lieut. J. E. G. McGibbon** showed a cinematograph film of the bronchi. He said that there had been numerous accounts of cinematography of the larynx, but he had been unable to find any records of similar pictures of the bronchi, and he thought that the present film was unique. Had it not been for the interruption caused by the war the film might have been more comprehensive, but, even as it was, it showed the possibilities of the film as a means of teaching and research, of making permanent records, and of observing the results of treatment. It might also stimulate others to obtain better results. It had taken two years to arrive at the present simple technique, and the results were largely due to the generous and skilled advice of Mr. Buckstone of Messrs. Kodak's Medical Department, and of Mr. Percy Phelps of Messrs. Mayer and Phelps.

JOINT DISCUSSION No. 1

Section of Comparative Medicine with Section of  
Epidemiology and State Medicine

Chairman—C. H. ANDREWES, M.D., F.R.S.

(President of the Section of Comparative Medicine)

[February 21, 1940]

DISCUSSION ON SALMONELLA INFECTIONS

**Sir William Savage :** Salmonellas are true parasites, and nowhere outside the animal body are they present as saprophytes, neither are they natural habitants of any animal intestine. This view is not universally accepted, and the prevailing view when I started my earlier work on this point was that members of the group are found as normal habitants of some animal intestines, but on examining animal organs and intestines with great thoroughness I found no evidence of their presence. It is well known that when examining series of animals, rats in particular and occasionally pigs, a small proportion are found to show Salmonella strains. But Salmonella infections in animals are common and though a proportion of survivors continue to harbour the bacilli, I regard it as reasonably certain that when found they are merely examples of the carrier state. There is an extensive literature upon the subject which strongly confirms this point of view. It is of great practical importance, for if Salmonellas are true parasites, without any saprophytic base from which they can be recruited, then we have an easier problem in the prevention of their distribution.

The second fact demanding consideration is the very large number of Salmonella types which have been differentiated mainly on serological grounds and often supported by minute cultural differences. To-day the work of Bruce White, Kauffmann, and many others has given us a reliable basis of classification. The White-Kauffmann classification is very helpful.

The facts seem to indicate that these serological types are definite entities and not mutable, and I know of no evidence which I can accept, that any one type can be converted into another. Many strains have been kept under artificial cultivation for twenty years or more without evidence of change in essential serological characteristics. Of course changes from *smooth* to *rough* or the specific and non-specific phases are recognized as mutable and do not affect the essential serological characters.

So far as tested, all members of the Salmonella group exhibit a considerable amount of cross immunity, but show some variability culturally.

A third feature of the group is their varying pathological manifestations. While in individual recorded cases members of the group have been found associated with an extensive assortment of pathological conditions, for practical purposes there are three groups of manifestations. These are attacks of the continued fever type such as we associate with enteric fever, acute gastro-intestinal irritation, such as is so common in food poisoning outbreaks, and general infections of the septicæmic type. These pathological manifestations, even with the same organism, are frequently not the same in man and in animals.

A fourth and general feature is their extensive activity as a cause of disease in man, animals, and birds.

The practical prevention of Salmonella infections turns very largely upon whether we can definitely associate type, hosts, and pathological activity. While there is neither completely distinctive nor constant pathological action, nor completely restrictive hosts, there is a large body of evidence which suggests that individual types have their selected hosts and usually their specific variety of pathological activity. Although rapidly growing our data is very incomplete and more investigation along these lines is desirable. Dealing with some of the better-known types the following roughly represents our present knowledge :—

Type	Hosts	Usual pathological action	
		Man	Animals
<i>B. paratyphosus</i> B	Man only	Enteric type	Unknown
<i>B. paratyphosus</i> A	Man only	Enteric type	Unknown
<i>B. aertrycke</i>	Many mammals and birds and man. Quite undifferentiated	Gastro-enteritis	General infection
<i>B. enteritidis</i> (Gärtner-Jena)	Wide distribution including man. Rats may be host of preference	Gastro-enteritis. Occasionally septicæmia	General infection
<i>B. enteritidis</i> (Dublin)	Calves and cattle mainly. Also man	Gastro-enteritis, septicæmia, continued fever	General infection
<i>B. suispestifer</i> I and II	Pigs mainly but also other animals including man	Gastro-enteritis or septicæmia	General infection
Paratyphoid C	Man only	Enteric type	Not known
Pullorum, Sanguinarium	Fowls	Unknown	General infection (fowl typhoid)

I am aware that there is some evidence that paratyphoid B has been isolated from swine on one or two occasions, and that in possibly one outbreak it caused illness of food-poisoning type, but the instances are so exceedingly rare that for practical purposes we must regard *B. paratyphosus* B as a purely human parasite causing paratyphoid fever only. *B. aertrycke* has on a few occasions set up septicæmia in man but never the enteric type. These exceptional deviations from the normal method of action do not invalidate the general picture.

In the Salmonella group we can discern at present three subgroups undefined or on their way to being specialized, i.e. the quite unspecialized types of which *B. aertrycke* is the typical example attacking with fine disregard any mammal, bird, or man, the fully (or nearly fully) specialized types with a definite pattern of pathological action and a restricted host such as *B. abortus equi* (horses only), *B. abortus ovis* (lambs only), *B. pullorum* (fowls only), *B. paratyphosus* A and B (man only), and the types on their way to specialization. *B. enteritidis* (Dublin) is a good example as it is particularly a calf and cattle disease producing organism with man as a variant because we will not protect our milk supply. The two *suispestifer* types are similarly mainly pig disease producers with rare extensions to man. Now that *B. enteritidis* is split up into serological subtypes it will probably be found that, like the Dublin strain, the distribution is more specialized. One type, "Essen", is particularly present in ducks, and there is evidence that one type of *B. enteritidis* has as its host of selection the rat.

It is tempting to speculate on the paths along which the different types have evolved. Bruce White (1926) has done this to some extent based on the serological make-up, but I am doubtful whether serological factors are a reliable guide. Taking a broader, possibly a less scientific attitude, we must regard the unspecialized types such as *B. aertrycke* as nearer the common ancestor. It is the usual tendency of parasites with host specialization to undergo modifications to fit themselves to the peculiarities of their hosts. For example an undifferentiated *aertrycke* cannot establish itself as a human parasite; it is either swept out by the violence of its

activities or, if it does invade, it kills its host. *B. paratyphosus* B, so allied serologically, suggests itself as a variant which has adapted itself for a human host by shedding its irritant properties allowing it to stay sufficiently long in the lymphatic tissues of the intestine to penetrate into the lymph and blood-streams and set up a true infection.

It is of interest, and possibly of importance, to note that what I call the types on the way to specialization seem to be types which often set up the septicæmic type of disease. The two *B. suipestifer* types illustrate this point. Nabarro, White, Dyke and Scott (1929) cultivated *B. suipestifer* I from the joints of two children suffering from arthritis. Kuttner and Zepp (1932) describe seven cases, all in children, the exact pathological conditions not being clear, but all of septicæmic type in which *B. suipestifer* II was isolated by blood culture. Of these 11 cases 10 recovered. Kuttner and Zepp (1933) describe four additional cases in which *B. suipestifer* II was isolated, i.e. bronchopneumonia, pyarthrosis, purulent arthritis, septicæmia, cystitis, &c. There are other cases in the literature. Septicæmia manifestations are also not uncommon with the Dublin strain which can also set up continued fever.

Quite one of the most interesting problems raised by the differing activities of Salmonella types is why closely allied types behave so differently in the animal body. *B. aertrycke* and *B. paratyphosus* B give the most striking example. The paratyphoid strain for man is purely an invasive organism with slight irritant properties, while *per os* it is non-pathogenic to mice or other animals. *B. aertrycke* in man acts almost invariably as a gastro-intestinal irritant, and when it does invade the blood-stream usually kills the patient, while for mice and other animals it sets up disease with general infection when fed. Bruce White and I (1929) studied the problem on the supposition that *aertrycke* possessed some undefined irritant properties not possessed by the paratyphoid strain. We carried out numerous experiments along many different lines. While we could demonstrate sometimes this irritant action we never obtained conclusive evidence that it was present more strongly in the types specially causing gastro-enteritis in man. The insensitiveness of the animal gut rather baffled us. It is possible, however, that these striking differences in mode of action are more associated with differences of host response. *Aertrycke* is an invasive organism for laboratory animals, the irritant action being relatively ineffective in the animal gut. Florey (1933) has carried out some investigations which are suggestive, dealing with the invasion of the intestinal wall by *B. aertrycke* in guinea-pigs. There was a large exudation of phagocytic cells (mainly polymorphonuclear) into the gut lumen, and these ingested large numbers of the bacilli which could be observed in all stages of digestion. Some of the epithelial cells were damaged, desquamated, and with the stroma laid bare. Some of the bacilli appeared to penetrate between the epithelial cells, others were passing through the cells themselves, the latter showing no histological evidence of death. Using non-pathogenic bacilli there was no evidence either of phagocytosis by the epithelium cells or of passage between them, nor were the bacteria phagocytosed by migrant cells in the lumen. *B. paratyphosus* B is non-pathogenic by mouth to the guinea-pig, so presumably it would act similarly, while for this animal *B. aertrycke* is an invasive organism. If it were possible to investigate the process in man along these lines I believe these two organisms might show interesting differences of local reaction in the gut.

These various scientific considerations are of much practical importance, and I will illustrate this by a few examples. In Salmonella food poisoning outbreaks much time is wasted in looking for a human carrier on the analogy of paratyphoid fever. If we realize that *B. paratyphosus* B does not cause food poisoning and that the common food-poisoning types such as *B. aertrycke* do not set up chronic human carriers it would save much waste of time in investigation. *B. aertrycke* does not invade the blood-stream in ordinary cases but lodges in the intestinal tract. After

recovery it may persist for a few weeks, but being a lodger it is swept out. Persisting for a few weeks such recovered cases may of course initiate a fresh outbreak, and there is a number of examples of outbreaks so spread, but this is very different from spread from a chronic carrier and the possibility of a recent temporary carrier can usually be eliminated by a few inquiries. A chronic carrier state is associated with living bacilli in the gall-bladder or other internal organ, and I know of no instance of this in man with *B. aertrycke*.

As an illustration of the practical value of differentiation of type I will take outbreaks due to *B. enteritidis* (Dublin).

Jensen in 1913 described a paracolon bacillus as one of the causes of calf dysentery in Denmark, and Uhlenhuth and Hübener showed that most of the strains were Gärtner group bacilli. In California Meyer, Traum and Roadhouse (1916) described *B. enteritidis* as the cause of a severe outbreak of infectious diarrhoea in calves. Other observers have subsequently shown the close association of *B. enteritidis* and disease in calves. Bruce White (1929) differentiated "Dublin" as different from the ordinary *B. enteritidis*, his strain coming from a fatal case of continued fever in Dublin. After its differentiation it was possible to show that it was this type which was especially associated with calves and cattle. For example Bosworth and Lovell (1931) recorded two epidemics of acute illness in calves with high mortality, due to Dublin. Knoth (1936), examining meat from slaughtered animals in Leipzig, found that of 538 strains from calves 506 were Dublin types, and 17 out of 18 from adult cattle were Dublin. Bartel (1938) examined 1,690 *Salmonella* strains isolated from domestic animals commonly used for food. Of these Gärtner-Kiel (Dublin) formed 78.37%. This strain was isolated 1,324 times, i.e. calves 1,027, cattle 253, pigs 28, horses 10, sheep 6. In calves the common clinical type was enteritis, but in the 253 adult cattle 95 were chronic carriers and 158 were emergency slaughtered animals with inflammation or other pathological involvement. Hohn and Herrmann (1939) found the Dublin type particularly common in calves and cattle.

#### HUMAN INFECTIONS WITH THE DUBLIN TYPE.

Place	Reference	Particulars
Dublin	Bruce White, 1929	Pyelitis kidney and continued fever; single case
Aberdeen	Smith and Scott, 1930	Three unconnected cases of continued fever. All positive blood cultures. All recovered
Aberdeen	J. Smith, 1933	Three unconnected cases (two infants, one 5 years): one septicaemia and mastoiditis, blood positive, fatal. One gastro-intestinal disturbance, blood negative, recovery. One meningitis, fatal; bacilli in cerebrospinal fluid
Aalborg (Denmark)	Grimsted, 1923	About 95 cases of acute gastro-enteritis at Aalborg Hospital. No deaths. Vehicle milk. Diseased cow which died and <i>B. paracoli</i> isolated from spleen and udder. Same organism in faeces of cases
St. Pancras (London)	Ministry of Health Report, 1928	Cases 22, no deaths. Vehicle junket. Suggested that was locally infected but information indefinite. Dublin isolated from faeces of cases
Dundee, 1927	Tulloch, 1939	About 280 cases of acute gastro-enteritis, no deaths. Vehicle milk. Dublin type from faeces and from internal organs of a diseased cow
Wilton, 1936	Conybeare and Thornton, 1938	Over 100 cases of gastro-enteritis in children, no deaths. Vehicle milk. Faeces examined late and negative. Milk contained Dublin and this isolated from dung of cow with high titre
S. Africa, 1938	Henning, 1938	Ten natives ate sick calf under-cooked. All suffered from food poisoning and one died. Dublin isolated from fatal case.

Since the differentiation of Dublin it has been found to be the cause of human infections in a number of cases. No doubt a number of outbreaks due to *B. enteritidis*, especially from milk, were due to Dublin, but as the strains have not survived to be differentiated we have no accurate knowledge. Without making any special search in the literature the foregoing are the human outbreaks I have come across.

It is highly probable that the following two outbreaks were Dublin types. Newcastle-upon-Tyne outbreak of 1913 (Kerr and Hutchens, 1914). Acute gastro-enteritis, 523 cases, no deaths. Vehicle milk and the mixed milk contained the milk of a definitely diseased cow which died. *B. enteritidis* isolated from faeces of cases and from the organs of the dead cow.

The Aberdeen outbreak of 1925 (Kinloch, Smith and Taylor, 1926) comprised 497 cases of acute gastro-enteritis with one death, an invalid aged 85, vehicle milk. Here again *B. enteritidis* was isolated from vomit and faeces of cases, from the milk and from the udder and flesh of a diseased cow which contributed to the milk. For both outbreaks the final determination, whether the *B. enteritidis* is the Dublin variety, is lacking.

We have here a type which at present is in the stage of causing all three types of pathological change in man, but which for animals has fairly restricted hosts; in fact I have seen few records of its presence in any animals apart from cattle.

I have noted for a great many years the fact that the mortality in these milk-spread outbreaks was comparatively insignificant and never understood why, since I anticipated the *Salmonella* organisms would multiply in the milk and with this heavy dosage would cause deaths. The type determination of Dublin seems to supply the answer. It has gone so far on the road to specialization in the calf that its pathogenicity for other species is low. It seems very rare in other animals and in man it is definitely of low pathogenicity. The supestifer and the Dublin types seem to be singularly alike both in their specialization in animal hosts and their diversity of action in man with a low mortality rate, at least when they cause the gastro-intestinal complex.

I rather emphasize these points not only because for nearly thirty years I have stressed that we must look for our sources of infection in food poisoning to *Salmonella* animal diseases, but because they illustrate a fact I did not so readily appreciate, that effective control is greatly helped by clear ideas as to the differential distribution of the different types.

We have still a long way to go before we can effectively prevent the pathological manifestations of the *Salmonella* group in man and animals. A potent weapon is an accurate knowledge of the distribution in nature of the various types and of their specialized pathological activities.

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**Dr. R. Lovell** : Almost all domestic animals may suffer from infection with *Salmonella* bacteria. Many cases of infection are sporadic and more or less accidental. On the other hand one particular species of animal may be commonly affected with one special type of *Salmonella* and act as its host reservoir. These reservoirs are important, for the more we know of them the greater our control will be over the diseases. A further point concerns the importance of *Salmonella* diseases of animals, some of which are solely of economic interest, whilst others concern the public health because of the pathogenicity of the organism for man.

The well-defined *Salmonella* diseases of animals may be due to one member of the group, but in some cases more than one type plays a part. Examples of this may be cited : Abortion of mares due to *S. abortus equi*, abortion of sheep due to *S. abortus ovis*, bacillary white diarrhoea of chicks caused by *S. pullorum*, and fowl typhoid by *S. gallinarum*.

On the other hand the common *Salmonella* disease of ducks called in America "Keel disease" may be caused by *S. typhi murium*, *S. enteritidis*, and in America *S. anatum*.

The control of *Salmonella* infections involves a knowledge of the host reservoirs, and this is well known as regards some types. Fowls are the common host of *S. pullorum* and *S. gallinarum*, whilst that of *S. abortus equi* is horses, *S. abortus ovis* sheep, *S. enteritidis* var. dublin probably cattle, and the variants of *S. cholerae suis* probably swine. Conversely *S. typhi murium* and *S. enteritidis* are more cosmopolitan, the former having been found, in addition to man, in rodents, pigs, cattle, sheep, fowls, ducks, pigeons, turkeys, and horses, &c., the latter in man, pigs, cattle, ducks, rodents, &c. Our knowledge of the range of natural pathogenicity enjoyed by some species is increasing. Some of the less common varieties, formerly found solely in man, have now appeared as animal pathogens. The Senftenberg-Newcastle type was originally isolated from man, but recently caused disease in turkeys (P. R. Edwards, 1937); the Derby type, which caused food poisoning in man, was later associated with disease in pigs, and the Dublin type is now recognized as a common pathogen of calves. A similar story concerns the Oranienburg type which, when first isolated from man in 1930 by Kauffmann, was not known definitely to be a human pathogen, but it was subsequently isolated from cases of disease. In 1936 Edwards isolated it from an outbreak of disease in baby quail, and in 1939 Jungherr and Clancy isolated it from chicken. From a similar source they recovered *S. london* and *S. bareilly*, both of which had previously been recovered solely from man.

Recently dogs and cats have been incriminated in the spread of human disease. Magnusson (1938) and Gard (1938) have described *S. abortus canis* as a cause of abortion in a bitch and a paratyphoid-like disease in man, whilst Kauffmann and Henningsen (1938) also described a new type, *S. braenderup*, causing in man a severe gastro-enteritis lasting a week and the death of his cat with a bloody diarrhoea. Later these authors (Kauffmann and Henningsen, 1939) describe *S. glostrup* as a cause of gastro-enteritis in seven people and a dog. The infection was assumed to be common to the human beings and the dog. *S. paratyphi* B is usually considered as solely a human pathogen, but in a small outbreak of paratyphoid fever in Norway in which *S. paratyphi* B was isolated from the human cases and three carriers, a young dog was incriminated as the source. The evidence in favour was confined to a history of fourteen days' illness, with vomiting and diarrhoea, and recovery just before the

first human case, and the possession of agglutinins in its serum, whereas 23 normal control dogs had no such agglutinins (Caspersen, 1938).

To enlarge our control of these outbreaks of disease attention should be paid to the careful identification of strains isolated from animals in attempts to increase our knowledge of the host reservoirs of particular types. Fifteen years ago this was pointed out in the Preface, "Food Poisoning. A Study of 100 Recent Outbreaks" (M.R.C. 92, 1925).

"Progress on that side of the inquiry in which it was desired to deal effectively with the paths of infection through domestic or agricultural animals has halted, in spite of much effort, . . ."

Eight years later J. Smith (1933), writing on sporadic *Salmonella* infections in the Aberdeen district, observes: "The striking features of the investigation have been the number of individual cases and the lack of definite evidence of the source of infection."

Within the last few years attempts have been made with such cosmopolitan types as *S. typhi murium* to subdivide them either by biochemical means or by the determination of serological variants and so establish the host reservoirs. Continental and American observers claim, for example, that the types isolated from pigeons and horses lack the somatic antigen factor V. It is no doubt true that many strains isolated from these two hosts do lack that factor, and the variants have received such names as *S. typhi murium* var. storrs or var. copenhagen. Lerche (1939) found that this factor was most frequently lacking in strains from horses, pigeons, and pheasants. He also found that all of 30 strains from man, 20 from duck eggs, 6 from sheep, and 47/60 strains from horses actively utilized ammonia salts as a sole source of N, whilst 21/50 strains from pigeons and 3/5 from pheasants did not. The evidence submitted in his paper indicates that these subtypes are not sufficiently clear-cut yet to have any epidemiological significance.

#### ASSOCIATION OF SALMONELLA AND VIRUS INFECTIONS

*Salmonella* bacteria were so frequently found in cases of hog cholera or swine fever that the actual cause was considered at one time to be due to a *Salmonella*. If animals are harbouring *Salmonellas* then infection with a virus appears to enable them to proliferate. They are, however, not found in more than a proportion of cases—Uhlenhuth *et al.* 1908, found them in 44.4%—and although *S. cholerae suis*—or a variant—is the common *Salmonella* of pigs, others have been found.

In other virus diseases, e.g. a condition in silver foxes in Norway (Holth, 1938), *Salmonellas* appear in association with a virus and young foxes infected with a filtrate of diseased tissue will develop a disease complicated with *Salmonella*.

*Salmonella* may also be isolated from dogs suffering from distemper (Dalling, private communication), and periodically this occurs in a batch of dogs from the same kennel. A similar condition sometimes occurs in ferrets, which have been infected with distemper virus and their origin was suspected to be raw pigeons which were fed to them. There is little doubt if dogs or ferrets are harbouring *Salmonellas* they can be isolated from the tissues following artificial or natural infection, with distemper virus.

The position is comparable with that which occurs occasionally in laboratory stocks of mice and guinea-pigs. After extremely cold weather, or after the injection of any substance, a proportion of mice or guinea-pigs may die from *Salmonella* infection. It is recognized that in outbreaks of *Salmonella* infections in pigs there are important predisposing causes associated with food and hygiene. This statement does not imply that *Salmonella* bacteria are harmless saprophytic bacteria of the animal intestine, a view which is held by some authors.

## DIAGNOSIS BY MEANS OF AGGLUTINATION TEST

As there are proportionately a greater number of latent infections than overt ones in many of the *Salmonella* diseases of animals some serological test is necessary for their detection. The recognition of carriers and their destruction is effective in controlling bacillary white diarrhoea and theoretically it should be as effective in the control of *Salmonella* diseases of mammals. There are, however, two main difficulties encountered in the interpretation of the agglutination test :—

- (i) The complicated antigenic structure of the *Salmonella* group.
- (ii) The presence of normal or natural agglutinins or, in the case of man, agglutinins due to previous inoculation with a typhoid-paratyphoid prophylactic.

Although the antigenic structure of the *Salmonella* group is well charted as regards the major antigenic factors, there are minor overlaps which are not always apparent. Using sera prepared artificially in rabbits, particularly after only two or three injections of a killed suspension, these major differences and similarities in *Salmonella* are well marked. If one uses sera from natural cases of disease then the results are not so clear-cut and, without going into more detail, it is generally considered that minor antigens are the cause of this. Felix (1924) pointed out that somatic agglutinins produced during paratyphoid A or B infection are often more readily detected by means of a suspension of a particular strain of the typhoid bacillus than by the homologous organism. Cruickshank (1939) has recorded his observations with human sera and considers that the cross-relationships are due to minor antigenic components. He points out the danger of using bacterial suspensions containing the X antigen described by Topley and Ayrton (1924). This antigen frequently appears in suspensions prepared from old broth cultures or even when grown on moist agar, it is heat-stable and is not specific for the different types of *Salmonella*.

The possession of normal agglutinins in the sera of human beings is usually low, and not the cause of much confusion. In inoculated persons—and this applies to most Europeans living overseas—serological tests often give unreliable results. Boyd (1939) points out that the flagella agglutination test was given up in military laboratories in India because other febrile conditions produced anamnestic reactions in those inoculated with typhoid-paratyphoid vaccine. Beattie and Elliot (1937) consider it desirable to carry out repeated quantitative tests using both H and O suspensions and to establish an initial level of agglutinins in inoculated persons. In typhoid fever agglutinins for the "Vi" antigen appear early and disappear early, and are useful for diagnosis in the first few days of the disease. Boyd considers that by such a test one can detect permanent carriers of the typhoid bacillus as distinct from temporary carriers or convalescents. His opinion receives some support from the observations of Pijper and Crocker (1937), but its use is so far confined to typhoid fever.

Normal or natural agglutinins in domestic animals constitute a much greater problem because of their greater frequency and higher content. Agglutinins are observed for "H" and "O" antigens, though in general the former are more frequent and both occur, more commonly, in older animals. These normal agglutinins do not appear to differ from immune agglutinins either in specificity as determined by the absorption test, or in heat resistance.

As in human disease, there is the greater difficulty in detecting carriers or those latently infected than in those which are ill or convalescent, and it is more often necessary to examine animal sera for evidence of latent infection. The normal level of agglutinins in the species of animal concerned must be established, and attention must be paid to the age and sex, for in some cases a higher frequency of normal agglutinins occurs in females. Different techniques are used in different laboratories, and the sensitivity of the suspensions used may vary according to the opacity and

method of preparation. Furthermore, differences may arise according to the strains used and the temperature and time of incubation of the test. When wishing to detect carriers, it is therefore advisable to test a number of or all the sera in the particular herd to establish the normal level. Daubney (1938) detects bovines, which carry *S. enteritidis* var. dublin, by an examination of the sera of whole herds, and Conybeare and Thornton (1938) used a similar method in detecting the cow responsible for an outbreak of gastro-enteritis due to this organism.

TABLE I.

	Age	<i>S. typhi murium</i>			<i>S. newport</i>		<i>S. paratyphi C</i>		<i>S. enteritidis</i>	
		Type	Group	"O"	Type	"O"	Type	"O"	"H"	"O"
Pigs—										
	8 m.	10	—	5	20	—	20	—	20	5
	8 m.	80	20	5	160	—	80	—	80	—
	7 m.	10	40	5	40	—	80	—	80	—
	7 m.	20	40	5	40	10	640	—	80	5
	7 m.	10	10	10	10	5	40	10	10	5
Cattle—										
	4.5 y.	80	10	10	80	40	160	—	160	40
	2.5 y.	40	10	10	80	10	40	10	320	20
	3 y.	80	5	5	40	5	40	—	40	5
	6.25 y.	20	10	10	40	20	40	—	40	20
	3.25 y.	20	20	5	40	20	10	—	10	10
Sheep—										
	6 m.	20	80	10	10	10	5	10	40	10
	6 m.	—	10	5	20	5	20	—	20	—
	6 m.	20	10	5	20	10	20	5	40	—
Horses—										
	12 y.	20	10	5	5	5	40	—	80	5
	16 y.	40	10	10	20	10	10	—	20	10
	18 y.	10	10	20	20	10	20	—	20	10
Goats—										
		160	20	20	20	20	160	—	40	—
		20	—	10	20	—	40	—	40	—
		80	20	40	40	20	20	10	20	20

Titres expressed as reciprocals of dilutions.

— = no agglutination at 1/5.

TABLE II.

		<i>S. abortus ovis</i>		<i>S. cholerae suis</i>		<i>S. typhi murium</i>	
		"O "	Type	"O "	Type	"O "	
Group I	(a)	10	40	20	0	—	
		20	40	0	0	—	
		20	20	—	—	—	
	(b)	10	40	—	—	—	
		10	0	—	—	—	
		0	0	—	—	—	
Group II	40	80	0	0	—		
	40	40	0	80	—		
	20	40	10	0	—		
Group III	320	2,560	20	160	40		
	160	160	40	160	—		
	160	160	0	80	—		

Group I (a) From slaughter houses.

(b) From selected clean flocks.

Group II Past infection (two years previously).

Group III Recent infection { Few days after abortion.  
Two to three months after abortion.  
Six months after abortion

Titres expressed as reciprocals of dilutions.

To illustrate the difficulties inherent in the test two tables are shown. Table I gives the actual results obtained with a random selection of sera from normal healthy

animals. They are typical and taken from the records which constituted a study on normal agglutinins published a few years ago (Lovell, 1934). Table II is similar in that it is a random sample and gives typical results obtained by Bosworth (1932-33) who studied the difficulties obtaining in the detection of sheep infected with *S. abortus ovis*. It shows that although agglutinins for *S. abortus ovis* are present in the sera of normal sheep they are present in greater quantities in animals which have recently aborted, and this is still apparent, though less so, after a period of six months. Without the experience of control sheep, however, the diagnosis by serological tests alone might occasion some difficulty especially in sheep which, though infected, have given no sign of active disease for some time.

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**Dr. W. M. Scott :** *Salmonellas in healthy pigs at slaughter.*—The evidence as to the frequency with which *Salmonellas* can be found in pigs at slaughter is conflicting. Lovell (1934) gives a table from which it appears that six bacteriologists in various countries (four in England) examined a total of about 700 pigs without finding a single *Salmonella*, whereas six others, nearly all German, examined about 900 with an average of 7.5% positive, the percentage varying from 0.4 to 8.5%.

In Uruguay, Hormaeche and Salsamendi (1936) examined pigs at slaughter by culture of pooled material (mesenteric glands and spleen) from 46 batches, each batch consisting of material from 20 pigs. They found *Salmonellas* in 22 of the 46 batches, identifying the 30 strains they isolated by careful serological tests.

In spite of the negative English findings, pig products are quite commonly suspected as the cause of the *Salmonella* form of food poisoning in this country. In the year 1937, for example, there were 11 outbreaks ascribed to the consumption of various forms of pig flesh, involving 300 persons and causing 7 deaths; in some, at least, of these instances there was no likelihood of extraneous contamination of the suspected food, hence the presumption that a primary pig infection was responsible. Accordingly an attempt was made to find out whether English pigs, slaughtered apparently in perfect health, were in fact so free from *Salmonella* infection as previous investigations had suggested. A large bacon factory in the West of England agreed to submit (a) mesenteric glands and (b) spleens, taken with aseptic precautions immediately the abdomen was opened, from pigs which on the usual careful post-mortem inspection appeared to be free from disease. To economize time and labour at the laboratory the glands from 10 pigs were pooled and, similarly, the spleens,

In making cultures, two glands from each pig, dissected free from connective tissue, were plunged for ten seconds in boiling water to destroy surface contaminants; all these were then snipped into pieces with scissors and ground up in a mortar with sterile sand. About an inch of spleen from each pig was similarly scalded, pooled, and ground up. It may be mentioned that washings of the jars in which the specimens were sent were also cultured for *Salmonellas* as a control for outside contamination but were always negative (with one exception). The resulting creams were (a) spread on a MacConkey plate, about 20 mgm. being used, (b) added in quantity of about 1 gm. to 50 ml. of peptone water containing brilliant green (1:150,000), and (c) heavily inoculated on a plate of Wilson and Blair bismuth-sulphite medium (Difco brand). After incubation overnight (b) was plated out from the surface film on to a MacConkey plate. Non-lactose-fermenting colonies with the general appearance of *Salmonellas* on the MacConkey plates and black colonies on Wilson-Blair were tested in drops of various *Salmonella* sera and those agglutinating picked off for fermentation and further serological tests.

Out of the 50 batches of pigs so examined, equivalent to 500 pigs altogether, no less than 20 yielded *Salmonellas*. In two cases the spleens alone were positive; in one both the mesenteric glands and the spleens gave *Salmonella* colonies, the glands yielding two different types (*cholerae suis* and Dublin), while the spleen gave *S. cholerae suis* alone. In the remaining 17 positive batches the glands alone contained *Salmonellas*. Assuming that each positive batch owed its infection to one pig only, except in three instances from which two different *Salmonella* types were isolated—in which cases two animals may be assumed to have been infected—there were thus, at least, 23 *Salmonella*-infected pigs out of 500, equivalent to 4.6% as a minimum.

Though the pigs in question had been drawn from a wide area, it was thought that the conditions in this area might be peculiar. A bacon factory in East Anglia was, therefore, next investigated in the same manner. Of the 50 batches, each of 10 pigs, 16 were infected. Only one yielded *Salmonellas* from the spleens, the glands from the same batch also being positive, but with a different *Salmonella* type. One batch of glands yielded two types of *Salmonella* (*cholerae suis* and Gaertner). Making the same assumptions as before, at least 18 pigs out of the 500 were *Salmonella*-infected, a percentage of 3.6. I suspect that this finding underestimates the real extent of infection, as it was discovered later that the brilliant-green solution employed for the selective culture in peptone-water had deteriorated; the proportion of successful cultures in this medium was much less than with the first batch and the difference may represent missed cases.

A remark on the relative success of the various cultural procedures may be of interest. In dealing with the first set the preliminary culture in the selective brilliant-green peptone-water was by far the most successful, 13 out of the 23 positives being obtained by it alone, the other two methods giving negatives. In three instances the Wilson-Blair plate alone was positive, and in one instance the direct plating on MacConkey alone. As just observed, in the second set the selective brilliant-green peptone water was less successful, only three being positive in it alone, while direct MacConkey plates were alone positive in eight instances and Wilson-Blair in two. In only one instance did all three methods give positives, so that there is an evident advantage in applying more than one method and preferably several. The great advantage of the selective enrichment in brilliant-green peptone water is that a large quantity of the suspect material can be inoculated.

Of the 24 *Salmonellas* isolated from the West of England, 5 were *S. cholerae suis*, 5 were *S. typhi murium* (aertrycke), 4 were *S. thompson*, 3 *S. newport*, 2 *S. enteritidis* (gaertner), 2 *S. london*, 1 *S. dublin*, 1 *S. reading*, and 1 not yet identified as a type.

Of the 18 *Salmonellas* isolated from East Anglia, 5 were *S. cholerae suis*, 4 *S. typhi murium*, 3 *S. thompson*, 3 *S. newport*, 2 *S. enteritidis* (gaertner), and 1 of the gaertner group but not identified precisely. All the strains of *S. cholerae suis* were of the

Kunzendorf variety, i.e. non-dulcitate fermenters, forming abundant  $H_2S$  and giving group-phase colonies only.

*Comment.*—All these pigs were passed as fit for human food and were in fact so used, without, so far as is known, producing any case of food poisoning. The fact that spleen infection was so rare among them suggests that infection of the flesh was also rare, if present at all, which may account for a large part of this innocuity. Only such food preparations as are made from offal (sausages, faggots, &c.) seem likely to be commonly infected, and it is such preparations which are most commonly suspected in outbreaks of food poisoning. Even in these a good deal of subsequent proliferation of the *Salmonellas* would probably be necessary to provide a sufficient infecting dose for food poisoning symptoms to follow. Nevertheless the presence of such specific pathogens in potential human food must give rise to some misgiving, and investigation as to the source of the infection seems definitely called for.

The fact that the mesenteric glands were almost solely implicated might suggest that the infection was of recent alimentary origin, i.e. that infected material had been ingested within at most a day or two, though this may be an unjustified deduction since little is known as to the length of time during which infected glands will continue to harbour living bacteria. It is customary for pigs to be subjected to as much as thirty-six to forty-eight hours' fasting before slaughter, during which period they are usually crowded together in lairs, searching among sawdust or litter for anything to eat. It is possible that at this stage they ingest material contaminated by an occasional pig discharging *Salmonellas* in its faeces. The possibility that the faeces of rodents might also provide such infection was considered, but no evidence of rat-infestation of the lairs in question was forthcoming.

Further observations (a) on the possibility of isolating *Salmonellas* from the intestinal contents of pigs at slaughter, and (b) on the effect of abolishing the period of pre-slaughter starvation were being undertaken when the inquiry had to be suspended.

[My thanks are due to Mr. C. H. Ducksbury, M.R.C.V.S., whose care in taking the specimens greatly facilitated their examination.]

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**Mr. R. F. Gordon:** *Salmonella* infections in domesticated birds are not rare even if one excludes *S. pullorum* and *S. gallinarum*, and during the past twenty years frequent references have been made to outbreaks of disease caused by *S. typhi murium*, *S. enteritidis* (gaertner), and *S. anatum*. The isolation from birds of *S. paratyphosus B*, *S. abortus equi*, *S. newport* and *S. senftenberg* has also been recorded.

Outbreaks of food poisoning in man by *S. typhi murium* and *S. enteritidis*, gaertner, have been described in which the source of infection was considered to be infected duck eggs. It has also been shown that infection of duck eggs with *S. typhi murium* and *S. enteritidis*, gaertner occurs, and that one source of infection is the infected ovary.

In the course of routine poultry diagnosis work during the years 1933–39 infection with *S. typhi murium* has been noted in 57 instances, and with *S. enteritidis*, gaertner, in 8. Although these organisms have been encountered with greater frequency from chicks, the most extensive outbreaks with the greatest losses have been observed in ducklings.

There was an extensive outbreak of disease in ducklings during the winter and spring of 1938–39, due to *S. typhi murium*. There were approximately 57,000 ducklings on the affected premises and the losses totalled 30%, with a daily mortality of over 1,000.

Control measures were directed along two main lines: the control of infection amongst the affected ducklings on the rearing farm, and the detection and elimination of carriers in the breeding stock. The hygiene on the rearing farm was of a low standard, and improvements in sanitation and hygiene met with most satisfactory results, the mortality falling from 30% to below 3%.

A routine blood test of the entire breeding stock was carried out for the detection of carriers of *S. typhi murium* and *S. enteritidis*, gaertner. At the first test approximately 21,000 blood samples from adult ducks were submitted to the agglutination test, and of these 4.6% gave a positive reaction. Second and third tests were carried out at intervals of three months, but the percentage number of reactors was not decreased and the routine blood testing was not successful in reducing the number of carrier birds. This was mainly due to lack of correlated effort on the part of the owners. It was noted that the percentage number of reactors increased with the age of the birds.

In an attempt to determine to what extent egg-transmission occurred, 113 birds, which had given a positive reaction to the agglutination tests, were brought to the laboratory for experimental work. This work is still in progress, but a brief summary of it to date is given. 79 of these carrier birds have so far been examined bacteriologically, and from these the corresponding organism has been recovered from the tissues of 39. In most instances the organism was confined to the ovary, which was the sole organ affected in 30 of the ducks examined.

A small breeding experiment was carried out, two pens being set up, one containing reactors to *S. typhi murium* and the other reactors to *S. enteritidis*. Fertility and "hatchability" were low, but all hatched ducklings were healthy and reared well without losses to 6 months old, and when blood-tested at the age of 4 months, no reactors were found. All unhatched eggs, in addition to over 1,000 eggs laid by these carrier ducks, have been examined bacteriologically, but in no instance has either of the organisms been recovered.

The most effective method of reducing the gross mortality was by improving the hygiene on the rearing farm, while the mortality was not appreciably influenced by the persistence of carriers in the breeding flock. The results of breeding experiments and the cultural examination of eggs laid by reactor birds, together with field observations, indicates that egg transmission was not the most important factor in the dissemination of infection, in spite of the fact that the organisms were recovered from the ovary of 50% of those reactor ducks examined.

Dr. J. M. Alston said that he was interested in Sir William Savage's finding that infections due to certain of the *Salmonellas* show clinical differences from one another. His own experience was among sporadic infections (in which it was often impossible to trace the source to food or to other persons), and he did not think that such clear differences according to the type of bacillus were then observable. Perhaps the severity of infection made the differences more obvious in epidemics and the more acutely affected people were the most noticed; at the same time there might be, even in epidemics, less severe infections resembling those seen in general hospitals. Dr. Lovell's paper showed the increasingly great complexity of serological diagnosis of the *Salmonellas* and because of this and the temporary absence of antigenic factors identification of strains was often uncertain. If the discovery of more and more strains continued, the scheme of their antigenic analysis might finally become completed with all the possible combinations of the known factors. There was most difficulty when types differed only by an extra factor in one of them, and therefore Dr. Lovell's statement was interesting that minor differences in strains obtained from animals in the same epidemic should often be ignored.

Dr. Scott's report of the presence of several types of *Salmonellas* in the carcasses of many pigs from different sources made caution more and more necessary in attributing infection to a particular source, especially for legal purposes.

**Dr. Harry Schütze :** Over 80 types of Salmonella have so far been listed. The H antigens, which are given alphabetic symbols, have already used up the alphabet and the last type has received the symbol Z<sub>15</sub>.

The O antigens have reached the number XXIX, this last symbol having been given to an interesting organism, *S. ballerup*, isolated in Denmark by Dr. Fritz Kauffmann, which in a personal communication he tells me contains Felix's Vi antigen, in addition to its own distinctive antigens, somatic XXIX and flagellar Z<sub>14</sub>. There are now 3 Salmonellas which have as part of their constitution the Vi antigen of Felix: they are *S. typhi*, *S. paratyphi C*, and now *S. ballerup*.

A possible candidate for inclusion in the group has recently been isolated by Dr. Schiff in New York. It contains a new H antigen but its somatic structure consists of O antigens I, VI, XIV, and XXV, severally contained in a number of Salmonella types and collectively identical with the somatic antigens of the South African strain, *S. onderstepoort*. The difficulty about its admittance to the group lies in the abnormality of some of its cultural reactions. For example, it is a late lactose fermenter, produces acid and gas in saccharose, and liquefies gelatin. It may be thought that these characteristics should bar the organism from the group, but one must remember that apart from the fact that the very marked antigenic relationship speaks for admittance, the possession by several authentic Salmonellas of similar irregularities nullifies to some extent the claim to its exclusion. There exists, for instance, a variant of *S. anatum* which is a late lactose-fermenter; the three types, *S. dar-es-salaam*, *S. schleissheim*, and *S. abortus bovis* liquefy gelatin, while *S. eastbourne* produces indole. It may be argued that the Salmonella group would be the most convenient pigeon-hole for such an organism.

**Dr. R. St. John-Brooks :** I should like to bring to your notice certain aspects of the Report on the work of the International Salmonella Centre in Copenhagen from January 1938 to June 1939, which I presented to the Nomenclature Committee of the Association at the Third International Congress for Microbiology, held in New York last September, in the unavoidable absence of the Director of the Centre, Dr. F. Kauffmann.

Dr. Kauffmann stated that 37 Salmonella Centres had been created in various countries, to all of which the requisite sera and cultures were sent, i.e. 50 sera and 100 cultures for each centre. Sera and cultures were also supplied to various other institutes gratis on request. At the time of writing more than 70 different serological types of Salmonella had been recognized. The centres referred to are situated in the following places:—

New York, Albany (U.S.A.), Lexington (U.S.A.), Toronto, Buenos Aires, Montevideo, Trinidad, Glenfield (Australia), Canberra, Onderstepoort (South Africa), Batavia, Saigon, Rio de Janeiro, Calcutta, Trivandrum (India), Shanghai, Mukden, Tokio, Kabete (Kenya), Stockholm, Oslo, Helsinki, London, Basel, Beograd, Budapest, Warsaw, Bucarest, Prague, Riga, Athens, Istanbul, Tel-Aviv, Siena, Utrecht, Paris, and Copenhagen.

It will be apparent that a world-wide intensive study of Salmonella problems is now being carried out.





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